This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

B 3 9015 00207 758 7
University of Michigan - BUHR

TRANSACTIONS

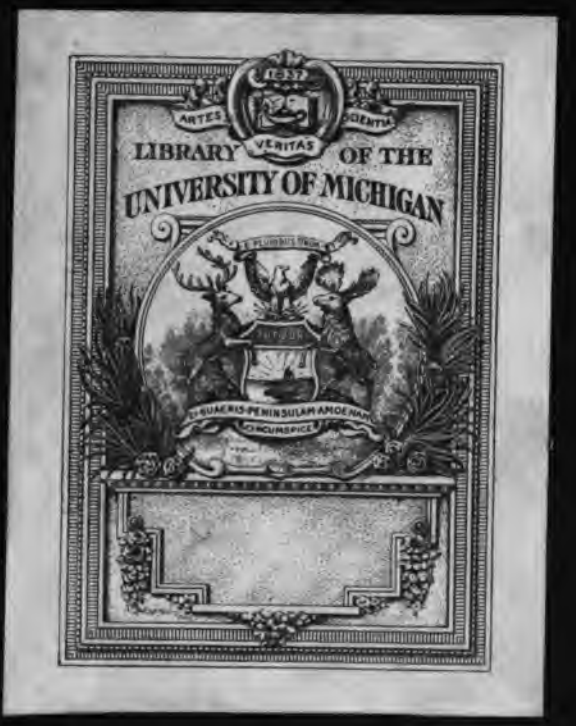
OF THE

BRITISH CONGRESS ON TUBERCULOSIS

FOR THE

PREVENTION OF CONSUMPTION

Digitized by Google


ARTES
SCIENTIA
VERITAS
LIBRARY OF THE
UNIVERSITY OF MICHIGAN
SUAE RIS PENINSULAM AMOENAM
CIRCUMSPICE

Digitized by Google

610.5
B86
C75
T8
1901

Digitized by Google

TRANSACTIONS

OF THE

British Congress on Tuberculosis

FOR THE PREVENTION OF CONSUMPTION. London. 1901

LONDON, JULY 22nd to 26th, 1901.

Patron:
HIS MAJESTY THE KING.

President:
H.R.H. FIELD-MARSHAL THE DUKE OF CAMBRIDGE, K.G.

VOLUME III.

REPORT OF THE MEDICAL SECTION.

EDITED BY
HECTOR MACKENZIE, M.D., AND SIR HUGH R. BEEVOR, BT., M.D.

REPORT OF THE PATHOLOGICAL SECTION.

EDITED BY
J. J. PERKINS, M.B.

LONDON:
WILLIAM CLOWES AND SONS, LIMITED,
23 COCKSPUR STREET, S.W.
1902.

Digitized by Google

LONDON:
PRINTED BY WILLIAM CLOWES AND SONS, LIMITED,
DUKE STREET, STAMFORD STREET, S.E., AND GREAT WINDMILL STREET, W.

Digitized by Google

09F12TR

TABLES OF CONTENTS.

SECTION II.

MEDICAL, INCLUDING CLIMATOLOGY AND SANATORIA.

PAGE

LIST OF OFFICERS OF THE SECTION 1

PRESIDENTIAL ADDRESS by—
Sir Richard Douglas Powell, Bt., K.C.V.O., M.D. 2

DISCUSSION ON CLIMATOLOGY 4

What Influence has Climate on the Treatment of Consumption, and how far can Cases be grouped for Treatment in certain Climates? . 4

Opening Addresses by—

Dr. Theodore Williams 4
Dr. Burney Yeo 15

Remarks in the Discussion by—

Sir Hermann Weber 21
Dr. Denison 24
Dr. Huggard 28
Sir John Moore 32
Professor Gram 32
Dr. Sandwith 33
Mr. Quarrier 34
Dr. Hobhouse 35
Dr. Crosby Walch 35
Dr. Exchaquet 36

Reply by—

Dr. Theodore Williams 36

PAPERS ON CLIMATOLOGY 38

A Query as to whether any special types of Pulmonary Tuberculosis and its complications are unsuitable for Treatment in the Climate of the British Isles. By Dr. Hichens 38

On Natural Immunity from Tuberculosis in Natal. By Dr. James Allen 39

Results of the High-Altitude Cure at the Sanatorium d'Hauteville (Ain), France. By Dr. Jonnart and Dr. Guetschel 51

De l'influence comparée des Climats et de la Résistance Individuelle dans la Tuberculose Expérimentale. Par MM. Lannelongue, Achard, et Gaillard 52

Über die Physiologischen Grundlagen der Hochgebirgskuren und deren Bedeutung für die Tuberculose. Von Dr. O. Amrein . . . 57

Digitized by Google

232769

PAPERS ON CLIMATOLOGY—*continued.*

PAGE

Tuberculosis in the Maltese Islands. By Dr. Zammit 74

Personal Experiences in Chest Measuring in Cases of Pulmonary Tuberculosis treated in High Altitudes. By the late Dr. Ruedi . . 74

Statistiques et Résultats de la Cure d'Altitude dans la Tuberculose Pulmonaire. By Dr. Stephani 77

Influence du Climat sur la Tuberculose observée dans l'Arsenal Maritime de Brest (Finistère). By Dr. Auffret 81

DISCUSSION ON THE THERAPEUTIC AND DIAGNOSTIC VALUE OF TUBERCULIN IN HUMAN TUBERCULOSIS 84

Opening Addresses by—

Dr. Heron 84

Professor Koch 93

Remarks in the Discussion by—

Sir R. Douglas Powell 96

Professor Osler 96

Professor Fränkel 97

Dr. Denys 97

Dr. C. Denison 98

Dr. C. Theodore Williams 99

Dr. McCall Anderson 100

Dr. Vicary Snow 105

Dr. E. C. Bousfield 105

Dr. Hamilton 105

Dr. Stone 106

Dr. France 106

Dr. Vivant 107

Dr. Squire 108

Dr. Moeller 108

Professor Sims Woodhead 110

Dr. Otis 110

Dr. Huggard 110

Professor Nolen 111

Dr. Heron 111

Reply by—

Dr. C. Theodore Williams 113

Professor Koch 114

PAPERS ON THE USE OF TUBERCULIN 117

On the Use of Tuberculin. By Dr. C. Denison 117

Sur le Séro-Diagnostic de la Tuberculose: Technique et Résultats. By Drs. Arloing et Courmont 128

Serum Diagnosis of Tuberculosis in Surgical Cases. By Drs. Arloing and Courmont 149

Recherches sur l'Agglutination du Bacille de Koch et le Cyto-Diagnostic dans Vingt-Quatre Cas d'Épanchements Séro-Fibrineuse de la Plèvre. By Drs. Widal et Ravant 150

The Agglutinating Reaction in Cases of Pulmonary Tuberculosis. By Drs. Horton-Smith and Armit 151

Frau Dr. med. Lydia Rabinowitsch 153

Digitized by Google

PAPERS ON THE USE OF TUBERCULIN—*continued.*

PAGE

De la Séro-Reaction comme moyen de Diagnostic de la Tuberculose des Divers Moyens de la Pratiquer. By Dr. Buard 153

La Prophylaxie de la Tuberculose Pulmonaire par la Connaissance de son Terrain. By Drs. Robin et Binet 157

The Hereditary Arthritic Forms of Tuberculosis. By Dr. Papillon . 165

Para-Tuberculous Hereditary Dystrophy. By Dr. Mosny 166

De la Transformation du Terrain Tuberculeux Hypoacide en Terrain Arthritique Hyperacide. By Dr. Bernheim 172

DISCUSSION ON SANATORIUM TREATMENT 174

Opening Addresses by—

Professor T. Clifford Allbutt 174

Dr. J. Kingston Fowler 186

Dr. R. W. Philip 190

A Report on the Results of Sanatorium Treatment in the United Kingdom. By Dr. Burton-Fanning 199

Remarks on the Discussion by—

Professor von Schrötter 205

Dr. John Dvorak 207

Dr. Jane Walker 211

Professor E. G. Janeway 211

Sir Hermann Weber 213

Dr. S. A. Knopf 214

Dr. W. Vicary Snow 215

Dr. Rosenthal 216

Dr. Burton-Fanning 218

Dr. Wolf Becher and Dr. Lennhoff 219

Dr. W. R. Huggard 226

Mr. J. C. Braine-Hartnell 226

Dr. Alfred Parsons 227

Dr. Howard Sinclair 227

Dr. F. Morin 229

Dr. C. K. Stone 230

Mr. G. W. Hambleton 231

Sir R. Douglas Powell 232

PAPERS ON SANATORIUM TREATMENT 234

A Plea for Seaside Sanatoria. By Sir Hermann Weber 238

The Classified Popular Sanatoria (Sanatoriums Populaires de Repartition). By Dr. Kuss 239

Traitement à l'Hôpital Pasteur du Havre. By Dr. Frottier . . . 247

L'œuvre de Sanatoriums Medicaux Français. By Dr. Verhaeren . . 247

Treatment in Institutions and in Open-Air Nursing Homes. By Dr. E. Symes Thompson 249

Remarks by—

Dr. Squire 261

Dr. Lasalle 263

Sanatorium et Villas Sanatoria. By Dr. Baradat 264

Digitized by Google

PAGE

ON THE USE OF THE RÖNTGEN RAYS 267

On the Use of the Röntgen Rays in the Diagnosis of Pulmonary Tuberculosis. By Dr. Hugh Walsham 267

Sur la Technique de l'Application des Rayons de Röntgen au Diagnostic de la Tuberculose. By Dr. Béclère 276

By Dr. Costa 284

Sur l'emploi de Rayons de Röntgen pour le Diagnostic Precoce de la Tuberculose Pulmonaire. By Dr. Bonnet Leon 285

The Importance of the X Rays as a means for the Early Diagnosis of Tuberculosis. By Dr. Espina y Capo 289

Difficulté de l'Interprétation des Images en Radiologie. By Dr. Mignon 294

Demonstrations on Cases of Lupus Vulgaris treated by Finsen's Method and by the X Rays; also results of other forms of Treatment. By Dr. Sequeira, Dr. Dore and Mr. Malcolm Morris, Mr. Startin, and Mr. Buck 295

MISCELLANEOUS PAPERS—

On the Value of the Rectal Temperature in Pulmonary Tuberculosis. By Mr. W. Braine-Hartnell 299

Some Observations upon Dietetics in Pulmonary Tuberculosis. By Dr. Noel Bardswell 309

Étude Anatomique et Expérimentale touchant l'influence de l'Alimentation par la Viande Crue sur l'Évolution de la Tuberculose Experimentale. Par MM. les Professeurs Cornil et Chantemesse . 312

The Treatment of Pulmonary Tuberculosis in Children by Raw Meat and Muscle Serum, according to the Method of Drs. Richet and Hericourt. By Drs. Albert Josias and J. Ch. Roux 317

Meat Albumin Dietary in the Treatment of Tuberculosis. By Dr. Forbes Ross 330

The Relation of Alcoholism to Tuberculosis. By Dr. Kelynack . . 334

Tuberculosis of the Heart. By Dr. H. Newton Heineman 344

Larynxtuberculose und Gravidität. Von Dr. A. Kuttner 353

The Principles of Treatment of Tuberculous Laryngitis. By Dr. StClair Thomson 356

Tuberculosis of the Eye. By Dr. Allen T. Haight 362

Devitalised Air Toxæmia—a Prime Cause of Tuberculosis. By Dr. C. Denison 374

Frequency and Significance of Tuberculosis of the Tracheal and Bronchial Glands in Miliary Tuberculosis of Children, and in Tuberculous Meningitis. Par MM. Haushalter and Fruhinsholz . 388

The *Rôle* of the Nasal Fossæ in the Prophylaxis and Treatment of Pulmonary Tuberculosis. By Dr. Mignon 390

The Use of Drugs in Pulmonary Tuberculosis. By Dr. W. R. Huggard 397

Remarks on Tuberculosis and its Treatment. By Dr. Baradat . . 401

The Treatment of Phthisis by Intra-Tracheal Injection of Large Quantities of Izal. By Mr. Colin Campbell 406

The Theory of the Action of Igazol. By Professor Vincenzo Cervello 415

Digitized by Google

MISCELLANEOUS PAPERS—*continued.*

PAGE

Treatment of Pulmonary Tuberculosis by Hypodermic Injections of Phosphate of Lime. By Dr. Angiulli 422

Du Cacodylate de Gaïacol dans le Traitement de la Tuberculose. By Dr. F. Barbary 429

Tuberculose et Sérums Antitoxiques. By Dr. Baradat 429

The Inhalation of Formic Aldehyde as an Aid in the Open-air Treatment of Pulmonary Tuberculosis. By Dr. Chowry Muthu . 434

Treatment of Pulmonary Tuberculosis by means of Formalin Intravenous Injections. By Dr. R. Maguire 438

On Time-Saving Methods of Treatment, with a Preliminary Note on the "Silver Treatment" of Phthisis by Intravenous Injections of Protargol. By Dr. Wm. Ewart 442

The Treatment of Tuberculosis by Urea. By Mr. A. H. Buck . . 445

Treatment of Anorexia in the Tuberculous by Persodine (Persulphate of Soda). By Dr. J. Hobbs 452

Sur un Nouveau Mode d'Excitation de la Peau dans le Traitement de la Tuberculose Pulmonaire—le Massage Pneumatique. By Dr. Ch. Breuillard 454

Treatment of Tuberculosis by High-frequency Currents. By J. A. Rivière 464

The Treatment of Tuberculosis by means of Electrical Currents of High Frequency and High Potential. By Mr. Chisholm Williams . 467

A Report on the Treatment of Tuberculosis by direct Introduction of Antiseptic Medicaments to the seat of the Malady through the pores of the skin by means of Static High Currents of Electricity of High and Medium Tension (Crôtte Method). By Dr. Leffingwell Hatch . 471

Report on the Treatment of Tuberculosis by Francisque Crôtte's Method. By Dr. Salivas 477

Report on some Experiences in the use of Francisque Crôtte's Method in the Treatment of Consumption. By Dr. Geisse 479

Conclusion of Proceedings of the Section 483

Digitized by Google

SECTION III.

PATHOLOGY, INCLUDING BACTERIOLOGY.

PAGE

LIST OF OFFICERS OF THE SECTION 485

DISCUSSION ON THE RELATIONS OF THE BACILLUS TUBERCULOSIS . . 485

Opening Addresses—

On the Relations of Tubercle Bacilli to other Bacteria resistant to Acids and to Actinomyces. By Dr. Moeller 485

The Morphological and Physiological Variations of the Bacillus Tuberculosis and its Relations. By Dr. Bulloch 494

Remarks in the Discussion by—

Professor Adami 503
Dr. McWeeney 504
Dr. Marmorek 504
Dr. Bousfield 504
Professor Kossel 505

Reply by—

Dr. Moeller 506
Dr. Bulloch 506

DISCUSSION ON THE CONVEYANCE OF TUBERCULOSIS BY MILK . . . 507

On the Infectivity of the Milk of Tuberculous Cows, the Bacteriological Diagnosis, and the practical value of Tuberculin in the extirpation of Tuberculosis among Cattle. By Dr. Lydia Rabinowitsch . . . 507

On the Infectivity of the Milk of Cows that react to the Tuberculin Test in the absence of Clinical Evidence of Tuberculosis. By Dr. McWeeney 514

Remarks in the Discussion by—

M. Nocard 515
Professor Adami 516
Professor Hamilton 518
Dr. Ravenel 519
Dr. Bousfield 519
Professor Sims Woodhead 519
Dr. McWeeney 521
Professor Bang 521

Digitized by Google

PAGE

DISCUSSION ON THE VARIETIES OF TUBERCULOSIS (MORBID ANATOMY AND HISTOLOGY) 523

Opening Addresses by—

Professor Benda 523
Professor Hamilton 530

The Interpretation of the Lesions caused by the Bacillus Tuberculosis. By Professor Hamilton 530

Remarks in the Discussion by—

Professor Sims Woodhead 536
Dr. Habershon 536
Professor Osler 539
Professor McWeeney 541
Sir Christopher Nixon 543
Professor Adami 544
Professor Muir 545
Dr. Bousfield 547

Reply by—

Professor Hamilton 548
Professor Benda 549

Paper on "The Comparative Virulence of the Tubercle Bacillus from Human and Bovine Sources." By Professor Ravenel 552

Paper on "Excretion Tuberculosis of the Kidney." By Dr. Walsham 583

Paper on "Some Experiments on the Temperature necessary for killing Tubercle Bacilli in Milk." By Professor Bang 592

Remarks in the Discussion by—

Dr. Ruata 598
Dr. Kossel 599
Professor McWeeney 601
Professor Delépine 601
Dr. Marmorek 602
Mr. Kingwell 602
Dr. Ruata 603
Dr. Ravenel 603
Professor Sims Woodhead 603

DISCUSSION ON MIXED INFECTIONS IN TUBERCULOSIS 604

Opening Papers by—

Mr. Foulerton 604
Professor Ophüls 614

Remarks in the Discussion by—

Dr. Bulloch 631

Digitized by Google

MISCELLANEOUS PAPERS—

PAGE

On Experiments proving the Infection of the Fœtus by the Bacilli of Tuberculosis. By Dr. Friedmann 632

Sur l'Immunité et la Sérothérapie de la Tuberculose. By Dr. Yabé . 635

A Study in Heredity: in its relation to Immunity and Selective Activity in Tuberculosis. By Dr. Maxon King 641

Étude sur quelques Tuberculoses locales: Formes cliniques et anatomiques particulières de la Localisation Tuberculeuse. By Dr. Clado 648

Effect of Exposure to Liquid Air upon the Vitality and Virulence of the Bacillus Tuberculosis. By Mr. Swithinbank 657

Steam-heated Railway Carriages and the Spread of Tuberculosis. By Mr. Brooke-Hunt 660

Rôle des Excreta Bacillifères dans la Propagation de la Tuberculose des Vaches laitières à l'Homme. Par MM. Boinet et Huon . . . 664

Digitized by Google

SECTION II.

MEDICAL, INCLUDING CLIMATOLOGY AND SANATORIA.

The Meetings were held in the Royal Medical and Chirurgical Society's Rooms, 20, Hanover Square, W., from Tuesday, July 23rd, to Friday, July 26th, 1901.

President.

SIR RICHARD DOUGLAS POWELL, BART., K.C.V.O., M.D., F.R.C.P. Lond.

Vice-Presidents.

Sir THOMAS LAUDER BRUNTON, F.R.S., M.D., LL.D., D.Sc., F.R.C.P. Lond.
W. WATSON CHEYNE, C.B., F.R.S., F.R.C.S., M.B., C.M.
Sir DYCE DUCKWORTH, M.D., LL.D., F.R.C.P. Lond.
Professor T. R. FRASER, F.R.S., M.D., LL.D.
Sir JOHN WILLIAM MOORE, A.B., M.D., ex-Pres. R.C.P.I.
J. E. POLLOCK, M.D., F.R.C.P. Lond.
Professor RODDICK, M.D., Montreal, Canada.
THOMAS P. A. STUART, LL.D., M.D., Sydney, N.S.W.
Sir HERMANN WEBER, M.D., F.R.C.P. Lond.
C. THEODORE WILLIAMS, M.D., M.A., F.R.C.P. Lond.

Hon. Secretaries.

HECTOR MACKENZIE, M.D., M.A., F.R.C.P. Lond., 34, Upper Brook Street, W.
Sir HUGH BEEVOR, Bart., M.D., F.R.C.P. Lond., 17, Wimpole Street, W.
R. W. PHILIP, M.D., M.A., F.R.C.P. Edin., 45, Charlotte Square, Edinburgh.
WILLIAM CALWELL, M.D., M.Ch., M.A., College Square North, Belfast.

Digitized by Google

ADDRESS BY THE PRESIDENT,

SIR RICHARD DOUGLAS POWELL, BART., K.C.V.O.,

Physician in Ordinary to H.M. the King; Consulting Physician to the Middlesex Hospital and to the Brompton Hospital for Consumption.

IT is my first duty to give a cordial British welcome to all our brothers in science who have come from distant parts of the Empire and from foreign countries to take part in this Conference and to discuss with us the many problems—social, hygienic, pathological, and therapeutical—connected with tuberculosis.

In this particular section the discussions will be mainly centred round three questions relating to the climatic and antitoxic treatment of tuberculosis and the use of sanatoria.

Tuberculosis is a disease, the specific lesions of which tend to heal, if we can secure them from contamination by putrefactive and suppurative organisms. This is the conviction that experience burns into our minds in our hygienic and therapeutic struggle with consumption.

Of the local causes that contribute to atmospheric contamination and so spoil climates we cannot here say much; they belong rather to the hygienic section. It is often disappointing to find a good climate marred by local impurities, and the vivifying power of the sun exhausted on organic *débris* in the atmosphere, so that little healing power is left for those who may have come long distances to seek it. How commonly does one find a habitation selected for the splendour of the climate in which it is situated, with, perhaps, an insecure cesspool or a foul farmyard in the near proximity to windward.

Apart from climatic cleanliness, however, and freedom from organic and inorganic dust, there are many other attributes of the atmosphere, such as degrees of sunlight, prevalence of winds, wetness or dryness of soil, range of temperature, relative humidity, rarity or denseness of air, and the like, which all enter into the calculation of the physician in advising his patient as to the climatic surroundings best for each.

It is scarcely conceivable that with the increasingly *al fresco* methods of our treatment, this important question of climate should not enter still more largely into our consideration, although some physicians are inclined to minimise their effects. We cannot fail to recognise the magnificent climates adapted to certain seasons that are afforded to those who have the means and the strength to seek them, in Switzerland, Egypt, the Riviera, and our Colonies and America. But in addition to what we may call climate in the gross we must look to climate in detail. I know an accomplished physician and scientific

Digitized by Google

horticulturist who, in his few acres of ground, can find a habitat for almost every plant that grows, and *we* may do much with the nooks and suntraps of these islands to make them suitable all the year round for that large proportion of the people who cannot go beyond them

There is still room, then, for profitable exchange of views with regard to climates and climatic adaptations, and especially in the choice of those best calculated to meet the needs of particular constitutional states or varieties of local lesion. We hope for an interesting discussion to follow the papers of our experienced colleagues, Dr. Theodore Williams and Dr. Yeo.

On sanatorium treatment, the position of which in this country is now thoroughly assured, a discussion will be commenced on Thursday by Professor Clifford Allbutt and Dr. Fowler, and a report has been drawn up by Dr. Burton Fanning giving much useful statistical information. We are much in need of a clear exposition of essential principles. Perfect asepticity in sanitation, skilled alimentation, and controlled metabolism would seem to be the headlines of our solicitude with regard to this system of treatment. A predominant medical control, with a separate bureau of finance, based upon medical requirements, and an accurate record of cases and results to be registered for professional purposes of comparison and information, are great desiderata for all sanatoria. Perhaps a brighter sympathy might be instilled into the handling of consumption, both in sanatoria and in ordinary practice, if the few necessary sanitary precautions of a personal kind might be guaranteed with less parade of detail and a more due sense of proportion. I have never felt convinced that tuberculosis requires the temperature to be taken in a manner unusual in other maladies, nor that the consumptive need be encouraged to train as a gastronomic athlete. Gain of weight is not the best measure of improvement, and, as I have elsewhere maintained, an excess of weight beyond that normal to the individual is a disadvantage. On the point of scientific feeding and assimilation in tuberculosis we hope for much enlightenment from some of the papers to be read at this congress. A small kur tax might secure some good music or lectures calculated to amuse and instruct without fatiguing or exciting the patients, and thus serve to keep the mental and spiritual side of consciousness going amongst them. The people who pass through sanatoria to live again with their fellows have learned valuable material lessons how to manage themselves, but there is danger of some impairment of those higher qualities with which consumptives are often conspicuously gifted, and which are of great value in rendering them hopeful and patient, and, when needs must be, resigned rather than despondent under the affliction of ill-health.

Of the use of tuberculin in phthisis we shall be eager to hear what can be said by Dr. Heron and Professor Koch, who will open the discussion to-morrow. It has thus far typified the wave-like advance of therapeutic pathology, and has been a gallant effort in the direction of antitoxic treatment.

There are many other papers to be read on Friday more or less connected with these three great topics, and we hope to formulate some practical conclusions from the work of this as of other sections of the congress.

Digitized by Google

"CLIMATOLOGY"—"WHAT INFLUENCE HAS CLIMATE ON THE TREATMENT OF CONSUMPTION, AND HOW FAR CAN CASES BE GROUPED FOR TREATMENT IN CERTAIN CLIMATES?"

By C. THEODORE WILLIAMS, M.A., M.D., F.R.C.P.,
Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.

GENTLEMEN,—The questions we are invited to discuss to-day are:

1. What influence has climate on the treatment of consumption?
2. How far can cases be grouped for treatment in certain climates?

Both these questions have been the objects of my study for the last thirty-five years, and I will place before the Section some of the conclusions which a large experience has enabled me to formulate, omitting, except where it is absolutely necessary, the facts and figures on which they are based.

I.

In all ages has existed a belief that consumption could be successfully treated by change of air—either from town to country, or from inland to seashore, or from valley and plain to mountain—and such belief has been the result of favourable experience of these changes.

In some lands, like Peru, the natives can point to instances of arrest of consumption from a transference of tuberculous folk from the sea-level to the mountains, and as no special attention has in these cases been paid to dietary, habits, nursing, or sanitation, the arrest of the disease must be attributed to change of climate, and to change of climate alone.

Sea voyages have possessed a great reputation for the treatment of phthisis, and especially in this country, and it must be admitted that they offer a most effective way of applying the open-air system; for what patient undergoing sanatorium treatment can obtain the amount of fresh air which comes into contact with the body and lungs of an invalid seated on the deck of an Australian-bound clipper or steamer as she speeds through the waters? Doubtless this is the cause of the improved powers of digestion and assimilation which are so marked a feature in sea voyages, and of the large gain of weight which follows even where the larders are indifferently stocked. A few years ago sea voyages were the commonest recommendation for consumptives, and we all probably can remember cases of striking advantage derived from them; but at present, though the vessels are better equipped, the food supply abundant and more varied, and the cabins better ventilated, sea voyages are rarely advised, for reasons which will be dealt with later.

Now, if we search the records of experience we shall find numerous and well-authenticated instances of arrest of tuberculous disease as the results not only of sea voyages, but of warm and cool marine climates; of dry, warm climates; and of mountain climates, respectively; and in fact it has been

Digitized by Google

proved that the arrest of tuberculosis has occurred in a variety of climates and under very different conditions.

Our object, however, is to determine under what climatic conditions arrest of the disease is most frequent, what are the real causes of such improvement, and how can they be successfully brought into play in other cases.

In all climatic treatment of course care must be taken to bring the patient fully under the influence of the atmosphere, and to ensure a strictly open-air life by day and by night, with complete hygienic and sanitary surroundings, whether in a sanatorium or elsewhere.

But all climates favouring an open-air life are not necessarily suitable for the treatment of consumption: tropical lands must be excluded, because there the appetite is diminished, the nervous and muscular energy depressed, excessive action from the skin induced, and requisite exercise prevented. Bacillary increase is probably also favoured. Very moist climates and those in which the sun is often obscured by mist and fog, even though mild, are undesirable, as sunshine is of the greatest importance to the consumptive. The climate which best fulfils the conditions for open-air treatment need not be a very warm or a very cold one, but should be dry and stimulating, with abundant sunshine, admitting of much exercise, and producing nervous and muscular vigour.

The Classification of Climates.

We must first classify climates before describing them, and though it would be possible to arrange them scientifically on the basis of meteorology, as I attempted in my Lumleian Lectures before the College of Physicians, for the purposes of this discussion a few practical groups will be preferable.

Classification.

1. Marine climates.
2. Dry, warm climates, partly inland and partly marine.
3. Mountain climates.

Group 1. *Marine Climates.*—Include (*a*) the British and Irish sea coast resorts; (*b*) the warm marine climates of Madeira, Teneriffe, and the West Indies; (*c*) Sea voyages.

Group 2. *Dry, Warm Climates.*—The desert climates, and those of the Mediterranean basin, differing in many particulars, but both characterised by warmth and dryness.

Group 3. *Mountain Climates.* — Characterised by the influence of diminished barometric pressure.

(1) Marine Climates.

First come the south coast stations on the British and Irish Channels, all more or less under the tempering influence of the Gulf Stream, and remarkable for a mild and equable climate, without extremes either in winter or summer, for a considerable rainfall, a large number of rainy days, and a prevalence of south-west wind.

Digitized by Google

The climate is bracing, and sometimes a good deal of sunshine is recorded, but on the whole it falls short in this particular of the Riviera and other southern localities, and the number of rainy days is a drawback. Some authorities object strongly to wind in a health station for consumptives, but I am in favour of its ventilating and purifying influence in the case of all diseases of germ origin, and protection by shelters can be successfully secured when wind is excessive. The great advantages of places like St. Leonards, Brighton, Worthing, Ventnor, Bournemouth, Torquay, Falmouth, Penzance, and Queenstown are their easy accessibility, their well-known medical staff, their excellent supplies of food and invalid comforts, and their being specially suited for the more weakly and advanced cases, where passive exercise is alone possible; and this last consideration will always have an unfavourable effect on their statistics of patients.

Patients can, as a rule, remain all the year round at the British and Irish south coast places, where they often become residents and form their homes, and this for open-air treatment is to be desired. At most of these stations are to be found a number of chronic tuberculous invalids who are firmly of opinion that they can live nowhere else. There are many, too, who have tried and benefited by other climates, but cannot leave England, or cannot afford to pass more winters abroad, and having achieved a certain degree of improvement find it more convenient to continue their treatment at home. The British climates are suitable for most cases of chronic pulmonary tuberculosis, and specially for the strumous forms.

The warm marine climates of Madeira, the Canaries, and the West Indies have not, in my experience, proved beneficial in consumption, and the statistics of Madeira patients which I published some years ago in my Lettsomian Lectures were very unfavourable, but the warm marine climate is specially suited to cases of catarrhal phthisis, that is where the tuberculous disease is accompanied by a large amount of bronchial catarrh, for it enables such patients, who shrink from cold, to tolerate and profit by an open-air life, which otherwise would be impossible for them. Such climates, and notably that of the Canaries, have been of some use in cases of laryngeal phthisis.

Sea Voyages.

Sea voyages have, perhaps, had the highest reputation of all, and certainly have yielded striking results, particularly when the voyages were to Australia and New Zealand round the Cape of Good Hope in clipper ships. The voyage occupied three months, and with one month's stay on land, and the return journey by the same route and not round Cape Horn, a good seven months' rest from worry and fatigue often put new life and energy into broken-down constitutions. Now the voyages are always in steamers, which curtail the time, and few go round the Cape, as the Suez Canal and Red Sea route is selected, a route fraught with danger to the consumptive from extremes and rapid vicissitudes of temperature during the voyage. Another objection is the lack of opportunity of exercise and of occupation, which in the case of young men often results in their spending too much of their time

Digitized by Google

in the smoking-room, drinking and betting on the run of the vessel. It is probably for these reasons, and also because other and better modes of treatment have been introduced, that sea voyages are not so often recommended as formerly. They are most beneficial (1) in cases of hæmorrhagic phthisis, where large hæmorrhages accompany small areas of tuberculisation; (2) in scrofulous or strumous phthisis, where lung disease is accompanied by strumous gland or joint affections; (3) in cases of chronic cavity, where the tuberculous disease is unilateral and quiescent.

In strumous phthisis I have repeatedly witnessed cavities contract and a gain of weight of from 1 to 2 st. occur during a sea voyage, and in patients with hæmorrhagic phthisis, where the hæmoptysis has been frequent and copious, I have known the patient completely exempt during a six months' voyage.

Of all the sea voyages, probably the best still is the Australian and New Zealand, as the tropics are passed through under the most favourable circumstances, and the temperature never rises above 80° F. or falls below 47°, except sometimes on the return voyage. Another good trip is the Brazilian voyage by Lisbon, Teneriffe, Rio de Janeiro, Buenos Ayres, and back, occupying two to three months; and another is the Royal Mail route to the West Indies, lasting six to eight weeks.

The evidence of Rattray and others goes strongly to show that the voyages should be partly in temperate climes and not only in tropical, for in the latter loss of weight often occurs.

Another great difficulty of sea voyages is ventilation of the cabin in rough weather. If the patient is fortunate enough to procure a deck cabin, well and good; but if it be deep in the hold and the portholes must be closed, the atmosphere becomes very impure, except in vessels fitted with a good system of extraction. The open-air life on deck during the day is excellent, and could the patient—as is sometimes done in hot weather—secure the same amount of air at night by sleeping on deck, the results would doubtless prove very good. Therefore, to ensure the success of a sea voyage, the patient must be certain of (1) good and abundant food, (2) proper cabin ventilation, also (3) that the cruise be principally in temperate climes and not in tropical.

(2) Dry Warm Climates.

Dry warm climates include the desert and the borders of the Mediterranean basin.

The Desert Climate.

The desert climate has as its best example that of the Egyptian desert, which is characterised by dryness and warmth, sunshine, and great radiation, and consequent great contrasts between day and night temperatures, also by its purity and asepticity of atmosphere. It is a climate which renders an open-air life by day and by night especially attractive, and life in a tent is not only possible but pleasant. The diminution of cough and expectoration is sometimes very marked, and catarrh as a rule disappears. The atmospheric purity has an excellent effect on secretion from the walls of cavities, and

Digitized by Google

hence the change in the character of the expectoration and the reduction in its amount. In my statistics of land climates, published some years ago, Egypt came out first in point of improvements, but my experience is that, although the tuberculous disease becomes quiescent, there is seldom proof of its absolute arrest, and I never witnessed the kind of results from the Nile climate that I do from high altitudes. The patients are relieved of distressing symptoms, but are not rendered more vigorous or more fit to resist the vicissitudes of the British weather. The Nile climate is suitable to elderly patients and to those who are prevented from taking exercise. Chronic pulmonary tuberculosis, especially with cavities, does well, and many advanced cases of consumption have in the desert lived for years, camping out in winter and in summer. The two objections to this climate are the Khamsin wind, which from the sand particles it brings is very trying, and the increase in the irrigation works and the great barrage systems, which are making the climate less dry, and therefore less fit for the treatment of consumption. Nevertheless, the climate of the Egyptian and Libyan deserts, from its dryness and asepticity, is probably the finest of all low-lying climates for consumption.

Climates of the Mediterranean Basin.

This group includes the Rivieras—French, Italian, and Spanish—the Mediterranean Islands, and Algiers and Tangier. These places are, as a rule, cooler, moister, and more stimulating than the desert, but they are liable to greater changes of weather if not of temperature. Compared with the British they are clearer and sunnier, with a good deal of wind, free from fog and mist, with a winter mean temperature of 8° to 10° F. higher, and with half the number of rainy days, and four or five times the number of cloudless ones. The French and Italian Riviera climate is as much abused as our own, because of undue expectations being entertained, but as an old observer of its elements I should say that its benefits arise from the combination of dryness and sunshine with cool winds and moderate warmth, which enable an invalid to sit as well as lie out in the open air most of the winter. When people abuse the mistral they forget that the dryness and fine weather come from that quarter, and that much of the bracing effect on the patients would be lost without this wind.

Another advantage of the Riviera is the coolness of the nights, though this is in many cases counterbalanced by the stimulating effect of the Mediterranean on the nervous system, which in some patients gives rise to insomnia, and in many exalts nervous sensibility. It is a great pity that the treatment of consumption is not more systematically carried out in this fine climate, and that sanatoria, or, at any rate, sanatorial treatment, is not established in every health station, as the results would undoubtedly prove successful, especially in cavity cases.

A large proportion of consumptive patients on the Riviera and elsewhere, as Sir Hermann Weber has well pointed out, consult the local medical man once, and no more, and then go their way—which is generally a very bad one—commit innumerable acts of imprudence, and thus lose all the advantages

Digitized by Google

which they might be expected to reap from the fine climate, and return home worse than they came. And thus time, money, and lives are wasted for want of timely submission to medical guidance and medical supervision. My own statistics of 210 patients, including a large proportion of excavation and bi-lateral affection cases, give a good percentage of general improvement (61 per cent.), but the percentage of local improvement is comparatively small, while that of deterioration is larger than is satisfactory, and not altogether to the credit of the Riviera.

However, in individual cases, and notably in the numerous medical men who have practised in the Riviera, we have plenty of examples of the arresting power of this climate in consumption. Where it does not arrest disease it certainly tends to prolong life, for I have known and published instances where comparatively advanced cases of consumption, which could not have survived our northern winters, have prolonged their lives for ten or even twenty years by passing their winters on the Riviera.

The class of cases which, according to my experience, benefit most by the Riviera are :—

1. Phthisis, in which inflammatory processes have played a large part in predisposing to the disease

2. Strumous phthisis.

3. Laryngeal phthisis.

4. Unilateral pulmonary tuberculisation rather than bi-lateral.

5. The large class of consumptives who either from extent of disease, or feebleness of circulation, or advancing years, are unable to endure the rarefied atmosphere and cold of the high altitudes. Most of these patients love warmth, and cannot take enough exercise, when the thermometer is below zero, to maintain it.

(3) Mountain Climates.

Mountain or high altitude climates vary according to altitude and latitude, but are all characterised by :—

(*a*) Diathermancy (Denison), by which the sun's rays are transmitted through the attenuated air, producing a difference between sun and shade temperatures of 1° F. for every rise of 235 feet.

(*b*) By their asepticity, as shown by the preservation of meat for long periods.

(*c*) By their physiological effects on the human body, as demonstrated by the tanning of the skin by the solar rays and chiefly by the ultra-violet rays (Bowles). The effect on the circulation is that it is at first quickened, and after six or eight weeks it is found to have slowed and to have gradually fallen to a rate below the normal. The breathing becomes deeper, the inspiration longer, and the expiration more complete.

The quickening of the circulation and respiration before acclimatisation is often accompanied by a great thirst and by a reduction in the blood pressure, and in the amount of urea excreted by the kidneys, but more carbonic acid (Marcet) and water are eliminated by the lungs. When acclimatisation is complete the urea appears in the urine in full quantity, and the blood pressure

Digitized by Google

increases. Accompanying the slowing of the pulse and respiration there is a dilatation of the thorax in various directions, causing increase in circumference from 1 to 3 inches at different chest levels, and there is increased mobility of the thoracic walls. All this arises from the greater physiological activity of the lungs due to the rarefaction of the atmosphere, and consequently their more complete development.

This thoracic enlargement is always more marked in those who take much exercise at high altitudes, and large thoraces are the feature of all mountain races, whether inhabiting the Alps, the Andes, or the Himalayas.

Statistics of Consumptive Cases at High Altitudes.

I will now give a brief account of 385 consumptives who have been submitted to high altitude climate for varying periods of residence between the years 1878 and 1900, and state the results arrived at.

Statistics of 247 patients were published in my Lumleian Lectures, and I have now added 138 more cases, as I have not complete notes of any more. I have classified these last on the same lines as the rest.

Sex.—Two hundred and seventy-five were males and 110 were females, the former preponderating over the latter as 5 to 2.

Age.—Table I. shows the ages of the two sexes when they started for the mountains, the average for the males being 29, and for the females 26. The majority of both sexes were between 20 and 40.

TABLE I.

Ages of 385 Consumptives before Residence at High Altitudes.

Age.	Males.		Females.		Total.	
	No.	%	No.	%	No.	%
10–20	27	9·81	18	16·36	45	11·68
20–30	136	49·45	60	54·54	196	50·9
30–40	82	29·81	28	25·45	110	28·57
40–50	28	10·18	4	3·63	32	8·31
Over 50	2	...	...	...	2	...
Total . . .	275	...	110	...	385	...

The average length of illness before climatic treatment was for the whole 385 patients, two years and one month; for the 275 males, one year eleven months; and for the 110 females, two years six months; which shows us that the cases were by no means incipient ones, but decidedly chronic before they tried the mountain treatment. Some had been ill for only a few months, some for 1, 2, 3, 4, 6, 7, 8, 10 years, and a few for even as much as 20 years.

Family predisposition was present in 143 cases, or in 37 per cent.

Hæmoptysis had occurred in 166 instances, or in 43 per cent.

Digitized by Google

Tubercle bacilli were found in the sputum of all these patients at some period of their history, excepting some treated before the era of tubercle bacillus discovery.

History and Nature of Cases.—In three of these patients there were malformations of the thorax, in one spinal curvature, in six tuberculous abscesses in various parts of the body, three had fistulæ in ano, one tuberculous disease of the testicle, two laryngeal tuberculosis, one laryngeal catarrh (not tuberculous), five had syphilis, in four there were cardiac lesions (one aortic and three mitral), one had dry basic pleurisy, and fifteen were cases of hæmorrhagic phthisis and four of catarrhal phthisis, the rest being examples of chronic tuberculosis of the lungs with a large preponderance of consolidation cases.

State of the Lungs.—I have placed on Table II. an extremely condensed account of the condition of lungs of these patients, which will convey to the members of the Section some idea, though necessarily an incomplete one, of their state at the time of taking up their residence at high altitudes.

TABLE II.

Influence of High Altitude Climate on 385 *Consumptives.*

Lung Condition.	Local Result.							General Result.		
	No. of Cases.	Arrest.	Diminished Physical Signs.	Improved including Arrest.	Stationary.	Worse.	Unknown.	Improved.	Stationary.	Worse.
Tuberculisation .	254	147	75	222	7	22	3			
Per cent. .	66	58		87	3	9				
Excavation . .	131	27	54	81	13	37				
Per cent. .	34	21		61	10	29				
Totals. . . .	385	174	129	303	20	59	3	87 %	1 %	12 %
Per cent. .		45·0	33·5	79·0	5·2	15·0				
Both lungs affected	139									
Per cent. .	36									
One lung affected	246									
Per cent. .	64									

The cases are divided into tuberculisation and excavation and single and double affection. The amount of tuberculous consolidation varied considerably, from that of an apex to a whole lobe, or even to a whole lung, and in many cases, as will be seen, there were consolidations in both lungs. Under excavation are included not only unilateral cavity cases, but also those where the opposite lung was also tuberculised. It will be seen that the tuberculisation cases numbered 254, or 66 per cent., and the excavation cases 131, or 34

Digitized by Google

per cent. Also that disease was unilateral in 246, or in 64 per cent., and bilateral in 139, or in 36 per cent.

Place and Length of Residence.—These 385 patients resided at high altitudes for periods varying from months to years, the shortest time being three months and the longest being nine years; the average being eleven and a half months, or roughly speaking, two winter seasons. In some instances, as in the Colorado patients and in many of the Davosers, the residence was continuous. The great majority of these patients resided at Davos (5,000 ft.), St. Moritz (6,100 ft.), and Arosa (6,200 ft.). Some were in Colorado (5,000 ft. to 8,000 ft.), and the rest, with one exception, in the South African Highlands (4,000 ft. to 5,000 ft.), up the Cape Colony, in the Orange River Colony, or in the Transvaal. One gentleman spent two years at Santa Fé de Bogota, in New Grenada, at an altitude of 8,648 ft. The patients lived on an average for eleven and a half months at an elevation of from 4,000 ft. to 6,000 ft. above sea level.

General Results.—173, or 45 per cent., of the patients completely recovered and retained their ordinary health; 77, or 20 per cent., greatly improved, and 54, or 14 per cent., improved. Placing all these together under the head of "improved," we arrive at a total of 334, or 87 per cent. of improvements.

The stationary cases numbered 5, or a little over 1 per cent., the worse 46, or 12 per cent., of whom 19 died under climatic treatment. The mortality at home will be considered later. About seven-eighths improved, and one-eighth deteriorated.

Gain of Weight was registered in 241 patients, loss in 29, and no change in 34. Gain of weight is not so marked a feature in the high altitude cases as in those treated in sanatoria, on account of the large amount of exercise usually taken; but a gain of from one to two stone, and even more, is not infrequent.

Local Results.

Most important are the results on the lungs. These are classified as:

(1) *Arrest*, signifying, in the case of tuberculisation cases, disappearance of all physical signs of disease, and in cases of excavation arrest signifies disappearance of cavernous sounds, and even, in some instances, of signs of consolidation, leaving nothing to be detected beyond deficiency of expansion over the whole side, and prolonged expiration above the scapula. In some cases not even these signs are present. These changes were always accompanied by the cessation of cough and expectoration, and the disappearance of tubercle bacilli from the sputum.

(2) *Diminution of physical signs* signifies that, while there is local improvement, there still remain some, though lessened, physical signs.

Worse indicates deterioration, either in the form of extension of disease, or softening or excavation, or both.

Stationary signifies no perceptible change in the local condition.

The 385 cases yield 174 absolute arrests—that is, 45 per cent.—and 129, or 33·50 per cent., of diminished physical signs. This gives a total improved of 303, or 79 per cent. The stationary cases number 20—that is, 5·2 per cent., and the worse 59, or 15 per cent.

Digitized by Google

In the tuberculisation cases we have 147 cases, or 58 per cent., of arrests, and 75, or 29·52 per cent., of "diminished signs," giving an improved total of 87·4 per cent.; the worse were 22, or 9 per cent., and the stationary 7, or 3 per cent.

The excavation cases yielded 27 arrests, or 20·6 per cent., and 54 with diminished physical signs, or 41·22 per cent., giving a total improved of 81, or 61·15 per cent.; 39 cases were worse, or 29·7 per cent., and 13 stationary, or 10 per cent.

These figures are very striking, and teach us clearly that tuberculisation cases do infinitely better at high altitudes than excavation ones, but that the latter make a very respectable appearance, and give a percentage of 61 per cent. of improved.

The numbers in the stationary and worse classes rather indicate that the climate has a decided influence, and promotes either improvement or deterioration.

Chest Measurements.

These were made in 133 patients, and showed an increase in circumference in 128, or 96 per cent. In five patients, who had pleurisy or extensive pulmonary fibrosis, no increase took place. The amount often reached three inches.

The foregoing account of these 385 patients only reports their condition up to their return from the high altitudes, and the question now arises as to what became of them subsequently, and as to what proportion relapsed, how many died, and in how many the disease remained in a state of arrest.

My first patients were sent to high altitudes in 1878, and I have continued sending consumptives ever since, so that these cases are spread over a period of twenty-three years, and during this time a certain number of deaths must be expected, especially among the elder persons.

I have information about 329 out of the 385, but of 56 could glean on news. Of these 329, 87 have died, or 22·6 per cent. Doubtless also some of the remaining 56 have died, but as a large proportion were leading an active life when lost sight of, it would not be fair to take it for granted that all have perished.

In addition to the deaths, 20 patients were ascertained to have suffered a relapse, but to have again recovered. Of the remainder the great majority are known to be in good health, and to be active during this present month.

SUMMARY OF CONCLUSIONS AS TO INFLUENCE OF HIGH ALTITUDES.

My conclusions as regards the effects of the climate of high altitudes on consumption are:—

1. That the respiration of the rarefied atmosphere produces hypertrophy of the healthy lung and local pulmonary emphysema around the tuberculous lesions, giving rise in due time to thoracic enlargement.

2. That it is possible the arrest of tuberculous disease is at least partly due to the pressure exercised on the tuberculous masses by the increasing

Digitized by Google

bulk of the surrounding lung tissue, which, by emptying the blood vessels, promotes caseation and cretification of the tubercle.

3. That these changes are accompanied by general improvement in digestion and assimilation, the cessation of all symptoms of disease, the return of natural functions, by gain of weight, of colour, of nervous and muscular activity, and of respiratory and circulatory power.

4. That arrest of disease takes place in 58 per cent. of the tuberculisation cases, and great improvement in 87 per cent. That in excavation cases arrest occurs in 21 per cent., and great improvement in 61 per cent.

5. That the climate is specially beneficial in hæmorrhagic phthisis, and phthisis in which hereditary predisposition is strongly marked, and is well suited to chronic tuberculosis of the lungs in general, provided the extent of lung involved is not too large, or the disease accompanied by much fever.

6. That males and females seem to do equally well and to profit most between the ages of 20 to 30. Males over 40 and females under 20 benefit least.

7. That the climate is contra-indicated in acute phthisis, catarrhal phthisis, in laryngeal phthisis, in cases of phthisis accompanied by great nervous irritability in patients with double cavities, with fibroid phthisis, and in all patients whose pulmonary surface has been so much reduced from any cause that it does not suffice for complete respiratory purposes.

TABLE III.

Results of Different Climates Compared.

	No. of Patients.	Average Length of Residence.	First Stage.	Second and Third Stages.	Bilateral Affection.	Results.						
						General.			Local.			
						Improved.	Stationary.	Worse (including deaths).	Arrest.	Improved (including arrest).	Stationary.	Worse.
	Per Ct.	Months.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.	Per Ct.
High Altitudes . .	247	12·2	65·	35·	37·	83·4	2·02	14·57	42·5	75·5	5·3	19·1
Sea Voyages. . .	65	1·6 (average of voyages)	63·	37·	37·	77·		21·56	7·7	53·3	10·7	33·8
Riviera	210	9·	59·	41·	36·	65·	10·	24·80	5·9	36·6	17·8	45·6
Home Climates . .	292	9·7	58·	42·	42·	63·7	8·21	28·	2·	38·9	20·	41·1

RESULTS OF DIFFERENT CLIMATES COMPARED.

The comparison of the results of the various groups of climates in my Lumleian Lectures, from which I extract the annexed table (Table III.), brought out the high altitudes at the top of the list, a position which they

Digitized by Google

have fully maintained, and especially as in most of the stations sanatorial treatment is also carried out, and if we want another example of the invigorating influence of the high altitudes we may find it in the determined resistance which our present enemies the Boers are offering to our brave army, for the Boers are a pastoral race, who have been reared and passed their lives at an altitude about 4,000 feet above sea level. Their chests are broad and deep, their frames are wiry and active, and they possess great powers of endurance, and though race may have something to say to the causation of their stout resistance, climate has still more.

In conclusion, I would say that, in my opinion, while not undervaluing the advantages of the hygienic treatment of consumption, a most potent weapon is to be found in climate, not only as proved by our results, but because of the invaluable assistance it renders to hygiene and medicine.

DR. I. BURNEY YEO, *Consulting Physician King's College Hospital.*

MR. PRESIDENT AND GENTLEMEN,—I shall endeavour to avoid, in the few remarks I have to make, going over the same ground as that so fully and ably traversed by Dr. Williams. I shall not, therefore, say anything further as to the classification of climates or the enumeration of their main characteristics. What Dr. Williams has said on these heads will be generally admitted.

The objects of treatment by climate in cases of pulmonary tuberculosis seem to me to be the following:—

(*a*) To arrest catarrhal conditions of the air passages.
(*b*) To improve nervous and circulatory tone.
(*c*) To increase the activity of the digestive functions, and thus stimulate nutrition by promoting the desire, and increasing the power, to take exercise.
(*d*) To raise the *moral* tone—by no means an unimportant matter—by affording a clear, bright, and cheerful environment.
(*e*) To diminish, by its asepticity, bacterial activity.

It must be a question for consideration whether so-called "open-air treatment," without regard to suitable climatic conditions, will do all this.

It should be our object, when practicable, to place the consumptive patient under conditions and in circumstances where, *without risk or injury*, he may obtain the most complete and perfect aeration of the lungs possible.

If you place a feeble catarrhal patient in the open air in a damp and cold climate you will risk an increase of the catarrh, and this will diminish pulmonary aeration by blocking up the air passages.

The modern "open-air" treatment is only "new" in its manner of carrying out this idea of hyper-aeration of the diseased lungs, and we must be especially careful in applying it to avoid the risk of aggravating catarrhal

Digitized by Google

conditions. This, I think, has now been fully admitted by some of the most strenuous advocates of "open-air" treatment *per se*.

The recommendation of a long sea voyage as a cure for phthisis doubtless had its origin in the idea of pulmonary hyper-aeration. It was an early form of "open-air" treatment, but with grave drawbacks and risks to which I shall immediately refer.

It may be interesting to mention that between two and three centuries ago Sydenham seems to have had in his mind also this same idea of hyper-aeration of the lungs in the treatment of consumption. He says, in "A Short Treatise of a Consumption":—

"The best remedy hitherto discovered . . . is riding sufficiently long journeys on horseback, provided this exercise be long enough continued. . . . For, in reality, the Peruvian bark is not more certainly curative of an intermittent fever than riding is of consumption."* And again, "But the principal assistant in the cure of this disease is riding on horseback every day, insomuch that whoever has recourse to this exercise in order to his cure need not be tied down to observe any rules in point of diet, nor be debarred of any kind of solid or liquid aliment, as *the cure depends wholly upon exercise*."*

Sydenham, in recognising in the treatment of phthisis the value of increasing pulmonary aeration by long-continued horse exercise, clearly anticipated the modern idea of open-air treatment of this disease.

Now climatic treatment is essentially "open-air" treatment, and the appropriate selection of a climate must depend on the suitability of that climate to an open-air life in the particular cases we may have to deal with.

It is difficult to establish any precise and rigid classification of the cases best suited to particular places because in many cases, and especially in very early cases, and in quiescent chronic cases, with a limited area of local disease, the patients will do well and obtain arrest of the disease in a variety of places with somewhat different climatic conditions.

One of the most remarkable arrests of phthisis I have ever seen in an advanced stage, *i.e.*, with a considerable area of consolidation and excavation in the upper lobe of the left lung, occurred in London.

I first saw this patient between twenty and twenty-five years ago; she was then about twenty years of age. She had at that time a considerable cavity in the upper part of the left lung, with much surrounding consolidation. She had dorsal curvature of the spine, and she had mitral disease, with a tendency to cyanosis. She was then living in the country, and I lost sight of her for a few years. She then re-appeared as a married woman, settled in London. The physical signs were much the same. I continued to see her, at long intervals, generally on account of some circulatory disturbance. She had three or four children, and kept her husband's house till she died about two years ago from the consequences of the mitral disease and not from the phthisis.

Another of the best results that I have seen, in a case in a fairly advanced stage, first came under my observation about fifteen years ago, and she never

* Swan's translation of Sydenham's Works, pp. 665 and 672.

Digitized by Google

went further from home than Hastings. The disease in the lungs has been arrested for some years, and she now occasionally consults me for dyspeptic troubles.

Another case that I have watched for nine years—a case of so-called fibroid phthisis—a case of what has been termed the pleurogenic type, for she has had many attacks of dry pleurisy, in the causation of which I consider rheumatism has played some part. She has a cavity in the left lung, and the whole lung from time to time becomes catarrhal. She has had several attacks of hæmoptysis.

She has done well in a varietyof places; she has wintered on the Riviera, in Algiers, in S. Africa, at Orotava, at Montreux, in Cairo; but she will *not* go where she cannot get social entertainment, and for that reason she prefers Egypt. As to living in a sanatorium, you might as well ask her to live in a prison.

I mention these instances to show how difficult it sometimes is to draw minute and precise conclusions as to the suitability of different places from the results observed in individual cases. Many chronic stationary cases with fair *general* health travel about to different winter resorts in successive seasons and appear to benefit, more or less, in all!

Another difficulty in drawing reliable conclusions from published statistics is the tendency on the part of certain observers to use somewhat vaguely the terms "incipient phthisis" and "pre-tubercular phthisis."

I find many practitioners are in the habit of using the term "incipient phthisis" in the sense of "suspected" phthisis. In that case it expresses an opinion rather than a fact. If this practice is largely acted upon it must greatly detract from the value of the statistics obtained.

It would be better, I venture to suggest, to discard the use of the term "incipient phthisis," and use the more precise term "early phthisis" instead; and then in the preparation of statistics it should be stated on what observed symptoms and physical signs this diagnosis is founded. The term "pre-tubercular phthisis" is, I think, still more objectionable. In estimating the results of any form of treatment these terms, I would suggest, should not be used.

We are, I suppose, all agreed that early cases with a very limited area of local disease, with little or no fever, with integrity of the digestive functions, and in young and otherwise healthy adults, do well and are frequently cured in a variety of climates, provided they live a perfectly hygienic, open-air life. They recover probably more speedily in altitude climates than elsewhere. Now it is only to the more vigorous of this group that I consider a sea voyage may be of use, and then only to those who have a liking for the sea. The value of the ocean voyage has been, in my opinion, greatly over-estimated and its serious drawbacks insufficiently realised. I have had occasion to know that many practitioners have advised a sea voyage as if it were a universal remedy for consumption, just as the "open-air treatment" is now prescribed, and dire have been the consequences.

The public love *unqualified advocacy* and universal remedies. We should be careful not to yield to the same tendency, and remember that our *rôle* is

Digitized by Google

to *discriminate*. I should like to read you a few lines about a sea voyage from the pen of one who was very eminent in his own branch of science:—

"The principal objection to persons in delicate health undertaking a long sea voyage is the *uncertainty* about the influences to which he or she may be exposed. While on land the traveller is to a great extent his own master, and has power to control the surrounding conditions. He may regulate the day's journey, according to strength or inclination; he may linger in such places as have agreeable associations and environment, he may hasten over those of an opposite character; but when once embarked upon a voyage—whether he finds himself crowded in a dark, close cabin, with two or three uncongenial companions, lying on a narrow, hard shelf, port-holes rigidly closed, and the atmosphere he breathes poisoned by noisome odours, of which the sickening smell of the oil of the engines is one of the least objectionable; the rain pouring on deck, making escape from his prison, even for a few minutes, impossible; when he feels he would give all his worldly possessions for a breath of pure air, or a few hours' cessation from the perpetual din of the engines within and the waves without—he is perfectly helpless; he must go through it day after day and night after night, until the weather changes or the voyage is ended."

This was the estimate of a sea voyage written by the late Sir William Flower.

Another brief estimate of the sea voyage by a consumptive physician who tried it, and who was one of my own clinical clerks, is as follows:—

"Any slight advantage there may be as to the pureness of the air of the ocean is, I maintain, far outweighed by the manifest disadvantages to an invalid at sea—such as the close, ill-ventilated cabins, the draughty saloons, the weakness and straining caused by sea-sickness, the damp sea fogs, the enforced confinement below during bad weather, and the great trial to an invalid of passing through the tropics."

Sir Andrew Clark, who had himself been in the Navy, remarked in 1890: "For my own part, when I now review the experience of the effects of sea voyages upon young invalids, I am more strongly impressed than ever I was before with the necessity of extreme caution in prescribing them."

Another point in which I conclude we shall all be agreed is that *decidedly advanced dyspnœic cases* should not, as a rule, be sent far from home, more especially if there is any pyrexia.

It will, however, occasionally happen that such patients amongst the upper classes insist on change, and get very restless at home; and they often rally a little on removal to some bright resort.

Of climates in which I have known distinct benefit follow in such advanced chronic cases, I would especially cite Madeira and Malaga. These advanced cases are generally associated with bronchial catarrh, and the warm, equable, and moist climates of the places I have mentioned soothe the cough, favour expectoration, and allow of the patient being much in the open air. One, a hæmorrhagic case, did well at Malaga, and another, complicated with albuminuria, passed the last two years of his life in comparative comfort in Madeira.

Digitized by Google

The idea that formerly prevailed that a warm, moist, and equable climate was the best for consumptives had a certain foundation in the suitability of such climates to the advanced catarrhal cases. There was little idea of cure associated with these climates, because consumption was then regarded as incurable; but it was thought that they prolonged life, and made the slow process of dying less painful.

The quality of *equability* in a climate was at one time greatly overrated. Indeed we—nowadays—avoid an equable climate when seeking a cure for early cases of pulmonary tuberculosis. We rather seek a climate with a very wide diurnal range of temperature, if it is a dry climate, as the Engadine or Egypt.

Wide diurnal variations of temperature exert a bracing, invigorating, tonic effect—especially when they follow a certain regularity.

What renders our own climate so very trying at times is that, although very variable, the variations of temperature follow no regularity. We get a week or ten days of very cold, fine, dry weather and then, just as the organism is adapting itself to the dry external cold, it changes and we get a spell of moist, wet, south-westerly winds, to be followed after a few days by a return of severely cold, dry weather, and so on. It is on this account that our climate can never be well suited to the out-of-door treatment of cases of catarrhal phthisis.

There is no great difficulty, then, in deciding what to do with cases at the very onset; we must be greatly influenced by questions of age, sex, temperament, occupation, social conditions, and constitutional tendency. They will get well in a variety of places with careful management.

Nor is there much room for hesitation as to what course it is best to follow in decidedly advanced cases. The progressive febrile cases are best in bed, with an abundant supply of fresh air. It is the moderately advanced case that calls for careful discrimination and is the most difficult to decide about.

It is now that the question of constitutional tendency comes into the foreground. Tuberculosis being an infective disease, attended with greater or less dissemination of toxic substances throughout the organism, we find, as we do with the attacks of other infective microbes, varying degrees of reaction, of susceptibility or infectibility, in different types of constitution.

It has been thought that the gouty constitution is antagonistic to tubercular infection. My impression is that the rheumatic constitution is so also, and that the latter is especially prone to develop the slow, fibroid pleurogenic form of phthisis.

Now this form is not, in my opinion, well suited to altitude climates. These cases do best in a warm and dry climate—such as the more protected resorts of the Riviera and the desert climate, as in Upper Egypt or Biskra. Setting aside this group, the high mountain resorts have doubtless the widest range of applicability to moderately advanced cases. There are, however, certain other cases that do not improve in these resorts. Early or moderately advanced cases, with manifest cachexia gastric disturbance and more or less fever, do not do well in these, nor, that I am aware of, in other resorts. A mild, marine climate perhaps suits these cachectic cases best.

Digitized by Google

Cases with laryngeal or intestinal complications should not be sent to the mountains.

Cases of much emphysema complicating tubercular infiltration, or tubercle invading emphysematous lungs will perhaps express better what I mean, are unsuited to altitude resorts.

Cases of this latter group are prone to attacks of almost continuous, and peculiarly uncontrollable, hæmorrhage, and are most unpromising. As might be expected, cases with renal complication do not do well in the mountains, and if albuminuria makes its appearance in such resorts the patient should be removed to a warmer climate.

A peculiar sensitiveness to cold is a very decided drawback to wintering in the mountains; for although the patient may be mending, so far as the local disease is concerned, he or she is always depressed and unhappy.

In our own country, in which sanatorium treatment is becoming justly popular, we must seek for suitable *local* climates in which the worst characteristics of our changeable insular climate are least felt, and where we can profit by what is best in it.

Nordrach is constantly referred to as a place in which consumptive patients get well in spite of the climate—but that place has great local advantages. It has a horse-shoe shaped screen of mountains, 1,400 feet high, protecting it from the north and east winds; it has adjacent pine woods where patients can take graduated exercise free from dust and protected from winds; it has an absorbent soil, and although it has a considerable rainfall, the rain runs off quickly and serves to keep the atmosphere pure and clean. Then it has also an elevation of 1,400 feet above the sea.

In England we have to consider the prevalence of fog and mist in many low-lying localities, the exposure to north-east and easterly winds in some, and to wet south-westerly gales in others.

What the consumptive patient most needs for his cure is a combination of climate and sanatorium treatment; for the patient, if left to his own devices, may make bad use of a good climate, while with skilful guidance in a sanatorium he may make good use of a bad one. Care without climate is better than climate without care.

The question put to us in this discussion is: "What influence has climate on the treatment of consumption, and how far can cases be grouped for treatment in certain climates?" The answer to the first part of the question will, I suggest, be that a suitable climate (*a*) relieves or removes catarrhal conditions accompanying the disease in a number of cases; (*b*) it raises nervous and vascular tone; (*c*) it increases muscular energy, and the ability as well as the desire for exercise; (*d*) by rendering an open-air life possible it increases the aeration of the lungs and diminishes the activity of bacterial agencies, one of the most essential conditions of arrest and cure of the disease; (*e*) it improves the tone and promotes the activity of the digestive functions, and so enables the patient to take the large amount of food which is needed to heighten his state of nutrition; and, finally (*f*), it improves the moral and mental state by surrounding the patient with a bright, cheerful and hopeful environment.

Digitized by Google

Then, as to the answer to the second part of the question, we may, I think, say—

1. That cases seen at the very commencement of the disease, and who are otherwise in good health, may be permitted a certain amount of choice in the selection of a climate, provided it allows of many hours being spent daily in the open air, and that they are placed under admittedly hygienic conditions.

A choice may be made from climates of altitude, the desert climate, the inland plateau of South Africa, the sea voyage for those with a decided liking for the sea, and suitably placed sanatoria.

2. For progressive febrile cases we should enjoin repose in bed or on a couch at home, in the best conditions practicable for the free access of air and sunshine to their apartments.

3. For advanced cases home is best, if the conditions of home-life are favourable, or the warm marine climates with cheerful surroundings, if home-life is unfavourable or change is urgently desired.

4. For catarrhal cases, warm, soothing climates like Madeira or Teneriffe are best.

5. For rheumatic or gouty cases of the fibroid or pleurogenic type, dry marine climates or the desert climate are most suitable.

6. For the so-called "scrofulous cases," if free from catarrh, fairly bracing marine climates; if with catarrh, mild marine climates should be prescribed.

7. For most other moderately advanced cases, with the limitations already mentioned, the climate of the high mountains, above the cloud-belt, is the most curative.

I have not made a "hæmorrhagic" group, because I do not think it would be a natural one. Every hæmorrhagic case must be, in my opinion, considered apart, and, if I may be permitted the phrase, dealt with "on its own merits."

PROFESSOR LANNELONGUE, *Paris*, read a Paper "De l'influence comparée des climats et de la résistance individuelle dans la tuberculose expérimentale" (*see* page 52).

SIR HERMANN WEBER, *London*.

Sir Hermann Weber alluded with envy to the vivid discussion of the preceding speaker, and expressed his admiration for the addresses by Dr. Theodore Williams and Dr. Burney Yeo. As to the remark by the latter, on the enthusiastic recommendation by Sydenham of riding, regarding this as an early instance of open-air treatment, he described the unexpectedly good results which he saw, between 1857 and 1865, in some poor German bakers and clockmakers, who became more and more ill while continuing their work in the bakehouses and in the confined workrooms, by recommending the bakers to push the bread-cars through the open streets, and the clockmakers not to work in the rooms, but to go about selling the clocks,

Digitized by Google

He said that these poor men came into the German hospital at the commencement of the disease, that they were improved for a time, but returned again and again, each time worse than before; and that at last he could see, on the one side, only certain death if they continued in their ordinary work, and, on the other, the chance—though slight—of recovery if they changed their indoor into outdoor work in all weathers. This plan led to much better results than he had expected, viz., about two recoveries out of three cases. This was open-air treatment in a haphazard way, but even this was better than the ordinary indoor treatment. One would hesitate now to give such advice, because one knows the beneficial influence of rest, but in exceptional circumstances one had to use exceptional means.

With regard to Dr. Williams' remarks about the use of marine localities, he (Sir H. Weber) found them more useful in scrofulous affections than in pulmonary tuberculosis, and thought that this was due to the high winds at most seaside localities, which, in many consumptive patients, caused catarrhal complaints. The injurious effects of high winds were shown in the distribution of phthisis mortality, especially in small islands, such as the Isle of Man, where the marine districts exposed to high winds manifested a much higher mortality than those more sheltered from wind; and this higher mortality was caused not only by the colder north and east winds, but also by the warmer south and south-west winds. He directed attention to a discussion at the Medico-Chirurgical Society some months ago, when Dr. Wm. Gordon, of Exeter, showed the great mortality in some South Devonshire valleys exposed to strong south-west winds.

Turning to the *warm, dry climates* of the *Western Riviera*, he remarked that his experience was not as good as he could wish, owing mostly to the patients avoiding medical control. The experience of the influence of sanatorium treatment at the Riviera was still in its infancy; but he had reason to regard the results of the treatment at the Gorbio Sanatorium, at an elevation near Mentone, under the management of Dr. Malibran, as satisfactory.

Amongst warm and dry climates, Egypt had always held a high place in the estimation of the public and the profession in the treatment of pulmonary tuberculosis, but hitherto the absence of medical supervision at most places had been a serious drawback, and had led in many hopeful cases to the saddest results. Excellent effects could, however, be obtained under satisfactory supervision, especially at Helouan and in the Nubian desert; arrangements for this purpose were in the course of development at Helouan, but were still quite absent in Nubia.

On another class of climatic resorts, the *high-altitude climates*, Sir Hermann greatly appreciated Dr. Williams' remarks; he had not himself been able to tabulate his experience during the last three to four years, but up to that time the effect of these climates on his patients had been better than that of any other climatic group or sea voyages, although he had repeatedly to deplore the deaths of most promising patients who, after many months of careful management, had almost entirely recovered and seemed to be on the point of being able to return home, when, in consequence of an act of imprudence, such as a mountain excursion, a long walk, or a tobogganing race, or a dance,

Digitized by Google

fresh outbreaks of the disease occurred, and led to death or to retardation of the recovery for several years. This was due, not to climate, but to want of supervision. He would, therefore, in future be inclined to divide his patients into such as were in sanatoria or under allied ways of treatment, and such as were out of sanatoria. Up to quite lately the majority of cases, especially of English, belonged to the latter class, and there were no sanatoria of the strictly high-level class excepting Dr. Turban's sanatorium at Davos, a smaller one at Arosa, and another at Leysin; but in the course of the last year another sanatorium had been established at Davos, high above the valley, on the Schatz Alp, by Drs. Spengler and Neumann, and others were in the course of erection. There would therefore be after some time the chance of comparing the results of the treatment of high altitude in and out of sanatoria.

There had been in former years an injurious custom at high altitudes, namely to send away the patients at the beginning of spring, on account of the melting of the snow, to lower and warmer regions, where the melting of the snow was over. On asking for the reason some said that the patients often caught colds during this period of the year, but with care colds can mostly be avoided; others said that the air was too moist. On this matter he stated that he had spent the end of March and first half of April of this year at "Les Avants," above Montreux. The melting of the snow was going on at a tremendous rate, so that within two days the snow melted from a depth of about 12 to 22 inches to almost complete disappearance, under the influence of one day of warm torrential rains, and of one of powerful sunshine. Les Avants being an official meteorological station, he had the opportunity of watching the hygrometer, and saw that it gave no indication of an abnormal humidity of the air, and that there was therefore no cause for sending away the patients on account of excessive humidity. In many cases the patients who were sent from the high elevations down to the Italian lakes and to the Riviera were attacked by various illnesses, and lost all or, at all events, much of what they had gained during the winter. Some of these patients he had attended together with the late Dr. Wilson Fox, who was known to all as a very judicious physician. On considering the circumstances they came to the resolution that they would only send those patients to the high Alps who promised that they would stay there over the period of the melting of the snow, and under this arrangement the patients fared much better.

On the point of *sea voyages* Sir Hermann Weber had been less successful than Dr. Williams, and would not recommend them to patients who were not originally endowed with strong constitutions, and were not good sailors. He endorsed the remarks of Dr. Burney Yeo. The salubrious effects of open air on the deck were sadly counteracted by the confined and impure air in the cabins, and even saloons. He especially mentioned that women, when ill on board ship, underwent terrible inconvenience and even suffering on long sea voyages, and ought to be sent only in exceptional cases; that several ladies, in consequence of their personal experience, had told him never to advise long sea voyages to invalid ladies, excepting they were perfect sailors and enthusiastically fond of life at sea.

Finally he expressed his opinion that, although he greatly valued the effect

Digitized by Google

of climates, especially elevated climates, he was convinced that in well-situated, well-arranged, and well-managed sanatoria in England, the prevention and cure of pulmonary tuberculosis could be very fairly accomplished, and that the effects of the climate of England would, in the majority of cases, compare well with those obtained in foreign more exhilarating climates.

DR. C. DENISON, *Colorado*,

would confine his remarks to the consideration of the high-altitude treatment of tuberculosis. First with reference to the relation of altitude to *outdoor life*, and second its relation to *deficient ventilation*.

1st. How does it happen that life out of doors is enhanced by altitude? The answer is, through its perfection in quality and its facility in use. All the attributes which go to make up the weather are favoured by a rightly chosen altitude. A choice must be made, for we have no intention of defending the extremes, either in Alaska, under the equator, or in Colorado. If the locality is appropriate the dryness of the atmosphere is likewise characteristic of the soil. The decreased cloudiness and long continuance of sunshine are important features, and the quality of the sunshine, through its diathermancy, is greatly enhanced. The stimulation of the cooling temperature with altitude, the increased heart action resulting, and the mechanical effect of the lessened atmospheric pressure upon the blood circulation, giving an onward push to the pulmonary currents and favouring a more complete capillary flow, especially with the thorax; the easy transpiration of vapour from the body, especially through the lungs, by means of the contrast in temperature between that of the body and the circumambient air, and the increased moisture-absorbing quality of the latter are important features. The free and constant motion of the atmosphere, the quick radiation of heat from all surfaces, and its heightened electrical state vivifying the air through its free static electricity and the resulting production of ozone. All of these, in fact almost every favourable quality of climate, are enhanced in their effect by an appropriate degree of air-rarefaction, provided the counter-condition is secured, namely, increased freedom of action and proportionate receptivity of the individual.

But this freedom of physical life, with its resultant bodily resistance to disease, especially to a dyscrasic one like tuberculosis, is only possible in its perfection when enjoyed out in the open. *Then* the cure of pulmonary tuberculosis as well as its hygienic prevention is established on a right basis.

2nd. How is it that the bad effect of deficient ventilation (which I believe is the main predisposing cause of tuberculosis) is increased by altitude? If there are impurities in the air, rarefaction would seem, on first thought, to thin them out and dissipate them. But it is not so much any defects pre-existing in the atmosphere which are dangerous, as it is the poisons we add by living in and breathing it. A barnyard with the sun shining on it may be a heaven of health compared with a closed rosewood palace with the influence

---

* Transactions American Climatological Association, 1900.

Digitized by Google

of the sun disbarred. It is the starvation of oxygen produced by air-rarefaction which magnifies the evil effects of deficient ventilation.

You may say, with Parkes' "Practical Hygiene" as authority, that the deprivation of oxygen is not felt ordinarily by healthy human beings till a loss of 14 per cent. is reached; and I may reply that the amount of oxygen which can be absorbed is limited any way, and that even when breathing pure oxygen saturation is only sooner reached than when breathing the ordinary air (Curtis). This does not, however, make much difference with the general proposition that for a given lessened barometric pressure a proportionately greater bulk of air must be breathed in order to get the equivalent amount of oxygen. In other words, to maintain the *status quo* of even deficient ventilation in the altitudes a proportionate additional space should be allowed for all sleeping and living rooms.

Imagine a hospital at sea-level, built, say, for fifty patients with an allowance of 2,000 cubic feet per person. Suppose this hospital were moved intact to a little more than a mile above the sea, say, between Denver and the eastern base of the mountains, where the barometric pressure is reduced to twelve pounds to the square inch—a loss of three pounds, or one-fifth. Without free ventilation that hospital must be enlarged to contain 120,000 cubic feet space in the patients' wards instead of 100,000 as before, in order to give them the same amount of oxygen on which to live.

The mechanical effect of the thinned air upon the functioned action of the heart and lungs could not compensate for the loss of oxygen in the closed rooms thus limited in size. In fact, by use the dead-air point would be sooner reached without ventilation than at sea-level. As the life of the air depends upon its free ventilation, that the vivifying influence of the sun may never be wholly shut out, so the hygienic prevention of tuberculosis comes to hinge upon this condition—free ventilation—and outdoor life is even more a *sine quâ non* of successful existence in high altitudes than it is at sea-level.

A consideration of the altitude of approximate immunity from tuberculosis confirms this contention. A study of the very few cases of tuberculosis which, with the advancing of our defective civilisation, do happen to occur in Colorado, should and does, as I will show, harmonise perfectly with this idea.

I do not suppose there is anyone so foolish as to claim an *absolute* immunity to tuberculosis infection for any elevation. On our side of the Atlantic Ocean much advantage has been sought to be gained by an unfair argument based upon the assumption that some one had claimed that there was "a *specific climate*" for the cure of tuberculosis. Such a climate would mean the impossibility of infection. I do not know of anyone who has made such a statement that there is "a specific climate." I cannot myself see why the conditions favourable for infection at sea-level might not be so accentuated at a high altitude as to produce the disease there.

In a State like Colorado, where so many invalids seek restoration to health, and where opportunities for contamination by the germ are so plentiful, why should not some of the many sources of infection prove effective? There is nothing in the climate to guarantee that a broncho or grip pneumonia, an imperfectly recovering typhoid fever, a suppurating appendicitis, or other

Digitized by Google

septic state long confining a patient under conditions which would be causative of tuberculosis at sea-level, will not have a like result in Colorado.

Yet the fact remains that tuberculous infection—reaching an advanced stage of the disease—is so rare an occurrence in Colorado as to be remarkable. Here, in the largest city in America, located at such an altitude, during the twenty-seven years I have practised medicine in Denver, the ratio of death from tuberculosis to all deaths, excluding those cases imported or contracted elsewhere, has been only from one-eighth of one per cent. up to less than four per cent.* During this same period cities in the eastern States have had a four or eight times greater ratio of tuberculosis to total mortality, exclusive of the migrating consumptives they have sent away to the south and to the Rocky Mountain regions, which greatly lessened their consumptive mortality and increased ours.

When one thinks—which I have seen—of an ignorant, third-stage consumptive from the East fastening himself as a visitor upon a poor family of eight, including little children, living in three small rooms, and he expectorating his myriads of bacilli upon the rough, unpainted floor, one wonders that there are not more cases of infection than there are, especially in a large city like Denver, where there are so many overcrowded small houses. Looking up and investigating the cases of tuberculosis probably originating in Colorado which I have myself seen or heard of during the past year, I find that they are all explainable in accordance with what has been said above. Two cases reported by other physicians in country towns seemed to be due possibly to other causes than known deficient ventilation.

One of these, an illegitimate young woman, age 22, with inheritance to tuberculosis on mother's side, never strong, and had broncho-pneumonia at 13, pleurisy at 17, pneumonia at 18, followed by night sweats and hæmorrhages a year or two later. Undoubtedly tubercular, but now under treatment and doing fairly well.

Another case, a young man of 22, is doubtful only as to the origination of his tuberculosis in Colorado, as his lungs were said to be "weak" when he came to this State at age of 12. Slept on ground floor in room 12 by 14 feet; had *la grippe* about two years ago, and never recovered entirely; was found to be tubercular in June, 1900.

My own cases of this class, four in number, are all doing well, especially when removed from the cramped, unventilated quarters in which they developed the disease.

(1) A young woman, age 18, born in Colorado, an undersized member of a family of seven, all of whom had "eczema" from doubtful, probably specific, inheritance on father's side; living in very cramped quarters near a mill ditch. The mother, a robust woman, says her daughter slept with her for nine months previous to her illness in bedroom 8 by 10 by 9 feet in size.

* This Denver four per cent. mortality is, according to a report tabulated by Dr. Wm. P. Munn when Health Commissioner, and for the six years 1893-98, inclusive. Two-ninths of these cases, said to have been contracted in Colorado, were classified as *meningeal tuberculosis*—a remarkable ratio and full of suggestion as to its cause. The record of mortality from tuberculosis for the whole State of Colorado has lately been found to be less than two per cent. of all deaths, that is of cases which were not imported.

Digitized by Google

This defective, neurotic, undeveloped girl, now 20 years of age, was found by me to be tubercular in third stage, June, 1899, though she claimed she had never been sick in bed.

(2.) An Italian woman, age 29, married nine years, and has five children, youngest one year old, not weaned from the breast when this patient was first seen by me in August, 1900. She was the wife of a former patient of mine who had miner's consumption. Five months previously this woman had a "severe cold," from which she had never recovered. The possibility of infection is admitted; but she slept with her two children in a close room, dimensions 9 by 10 by 8 feet, and was otherwise most unhygienic in the care of her person—in fact, had not taken a full bath for five months.

(3.) A poor sewing woman, age 37, undersized, spinster, lived in Colorado 21 years; always confined at home sewing. For three years has slept with her sister in a bedroom, size 11 by 9 by 9 feet, opening off from the kitchen. Had *la grippe* February, 1900, and in August the same year, cough and expectoration having continued since, we found bacilli in sputum.

(4.) A young woman of 27, married 6 years, clerk in a Denver agency office for three years, where there were eight others, all working in three adjoining rooms; two of these clerks were expectorating consumptives, at times using the waste baskets instead of spittoons. During all these three years she slept at night with her husband, in a room a consumptive had previously occupied, size about 8 by 10 by 9 feet, and it was on the ground floor and had but one window. This room was located on the north side of a small house which was shut in between two others. The tuberculosis was laryngeal as well as pulmonary, the sputum yielding T.B. germs in large numbers. It may have been a case of infection, as there were plenty of chances, but the condition as to deficient ventilation was certainly contributory. To get her out of town, Arizona was recommended, where by last accounts she was doing very well.

It is thus reasonable to conclude, I believe, that deficient ventilation is the key-note of the *cause*, as is outdoor living or perfect ventilation the key-note of the *cure* of tuberculosis. It is mainly an acquired disease, since Dr. Schreiber tried many new-born children with the tuberculin test, and "during the first week of life they did not once react;" that is, only a dyscrasic tendency is transmitted from parents to child. How variously but certainly this individual susceptibility is magnified by our faulty bringing-up and by our after-life constitutes the history of the acquirement of tuberculosis.

It is not acquired during a life out in the open, else our cattle, grazing on the western plains, would show the existence of the disease as they do not now. That acquirement, as is generally conceded by medical men, comes (as it does with monkeys and other animals) through domestication, with its too close confinement in unventilated rooms.

The best we can do, then, is to insist that the necessity of outdoor living must be appreciated, especially in Colorado, where the benefits of such a life are enhanced above what obtains in the lowlands: also let us insist that legislation take hold of this question of deficient ventilation, that this potent cause of this dyscrasic disease may be in a measure eliminated.

Digitized by Google

DR. HUGGARD, *Davos, Switzerland.*

THAT climate is not without importance in the treatment of phthisis is made evident by the improvement or deterioration that takes place in a large proportion of cases directly after a change of air. Now this might be ascribed to other influences, were it not for the fact that the improvement or deterioration is, as a rule, exactly as foreseen. Not an uncommon experience of mine is for a patient to come up to Davos contrary to medical advice, to lose ground there, and directly on being sent down to a suitable place in the lowlands to make rapid strides ahead. Or the case may be reversed. A young and comparatively vigorous patient sent to a warm climate fares badly there, and speedily gains ground on coming to the mountains. Instances of this kind are too frequent in my experience, and I am sure in the experience of all who have had the opportunity of observation, to permit of doubt as to the benefit to be gained from a suitably chosen climate in pulmonary tuberculosis.

The first point to recognise is that there is no *one* best climate for all cases. No climate has a monopoly of good effect. For every climate, too, there are unsuitable cases. In some instances climate is of comparatively little importance. That is to say, the patient will do well in any fairly good climate where he follows a suitable mode of life.

It is possible to select climate on wrong principles—to make the decision turn on points which, though important enough in themselves, are quite irrelevant for the purpose. The points that I specially mean are the presence or absence of a cavity and the duration of the disease—whether the disease is incipient or of long standing. Important as these considerations are in reference to prognosis, in reference to climate they are really as irrelevant.

Now what principles have we to guide us in the selection of climate for patients suffering from tuberculosis? The principles are simple enough, but the application of the principles will afford scope for the exercise of the utmost acumen and judgment. The problem is a threefold one. We must know the physiological or therapeutical action of the climates at our disposal; we must estimate the factors that determine the reaction or response of the patient to the various climatic influences, of which the chief is the demand for heat production; and we must decide on the correspondence or fitness, the relation between the patient before us and the climate we select.

Each of these aspects of the question may be looked at from a standpoint giving a broad view.

In regard to climate therapeutically, the most fundamental point is its action on tissue change; and its action on tissue change depends on its heat-abstracting powers. Now, the heat-abstracting qualities depend not on temperature alone, but on temperature in combination with the humidity of the air, relative and absolute, and the amount of wind. Climates can, therefore, be best arranged in series according to their physiological action. At one end of the series stand those climates whose heat demands are large; at the other end those climates whose heat demands are small. These climates may be subdivided according as the heat demand is regular or irregular in its variations. In warm climates the amount of moisture,

Digitized by Google

especially the relative humidity, is the chief circumstance determining the regularity of heat demand. If the relative humidity be high when the temperature falls a few degrees the water vapour in cooling becomes a sort of sponge to absorb heat from the body, radiation and evaporation at the same time being hindered. While heat is thus removed by the cooled watery vapour, the degree of cold may be insufficient to cause either the contraction of the surface vessels which would protect the inner organs, or such a response on the part of the organism as would lead to a more rapid development of heat.

In cold climates, on the contrary, the condition determining regularity in heat demand is the absence of wind, the humidity of the air, relative or absolute, being then of little importance. The amount of moisture is small even at saturation point. Further lowering of temperature, therefore, does not cause the chilling sensation that would occur from a similar fall in warm air of a like degree of relative humidity. Cold air—air below the freezing point of water—may, even when saturated with moisture, be regarded as dry in reference to the human body. But at such a temperature the slightest movement of the air greatly increases the demand for heat production. Equable climates are of necessity rather humid; and hence are amongst the most irregular and variable in their heat demands. Dry climates are never equable in temperature, though they may be regular—I do not say equable—in their heat demands.

High altitudes come into the group of climates having a demand for a high rate of tissue change. The hæmopoietic influence of high altitude—if it is a fact—is a favouring circumstance in the same direction.

Now let us look at the next aspect of the problem, the estimate of the patient's power of response to the demand for an altered degree of tissue change. The fundamental principle may be laid down that the best climate for a person is that where nutrition is at the highest level; and nutrition is at its highest level where the demand for tissue change—the demand for the production of heat—best coincides with the patient's power of response. The same climatic conditions do not act on all people alike. The response to heat and to cold differs enormously in different people. Patients may be classified according to their vitality or general vigour or power of resistance to cold. Whether a climate is bracing or relaxing or depressing depends mainly on the patient's power of response.

How is this power of response on the part of the patient to be determined? Apart from direct inquiry as to how the patient bears heat and cold, an examination of the muscular system and of the organic functions generally will enable us, in the large majority of instances, to come to a right conclusion. In some cases the point can be settled only by trial. Young people still in the period of growth usually respond well to a climatic demand for a high rate of heat production. The contrary is the case with old people as a rule.

In regard to the remaining aspect of the problem—the fitness of different classes of patients for different types of climate—certain prominent symptoms are of importance, though of importance quite subordinate in value to the broad principle just laid down.

The cold, dry climate characteristic of the Alpine resorts is suitable for

Digitized by Google

persons of robust constitution apart from their local disease; also for persons, though not robust, whose organs of nutrition and oxidation are sufficiently sound functionally to do much more work than they have been accustomed to at home. The lungs may be extensively damaged, but yet have reserve enough to respond easily to the increased demand.

Persons, on the contrary, of feeble circulation, with low digestive and assimilative powers, with small lung capacity, even though there may be but little damage present, will fail to respond to the cold, and so far from finding the climate bracing, will find it depressing in the extreme.

No stress whatever is to be laid upon the duration, extent, or stage of the disease pathologically. These considerations, though important from other points of view, are of little value in the selection of climate. Pyrexia, on the other hand, is always an important consideration in regard to climate. Generally a patient with much rise of temperature will be well advised not to undertake any long journey until the disease reaches a less active stage, even though at the end of his journey he should be in a more suitable climate.

The cases that get on well in the uplands of South Africa are in the main of the same character as those suitable for the Alps. A feverish tendency, however, may be considered to render a patient quite unfit for South Africa. Sub-acute cases, which commonly do well in the Alps, are thus practically excluded.

The innumerable high-lying resorts of the Rocky Mountains, of which Colorado Springs and Denver are the best known, answer well for the same class of cases as those indicated for the Alps and for the South African highlands. The American mountains are colder than the South African, and less cold than the Alpine resorts. They are more suitable for patients of low vitality than are the Alpine regions.

Wind and dust are amongst the chief drawbacks of nearly all American health resorts, and especially of those in the Rocky Mountains. The South African highlands suffer from the same plague. The Alpine resorts, owing to their covering of snow, are entirely free from dust during the winter; and in every case their comparatively sheltered position has been one of the factors that have led to their development.

One feature common to these three elevated regions—the Alps, the South African plateau, and the Rocky Mountains—is that the climate is a strong inducement to an open-air life.

The Mediterranean coast of France and of North Italy is marked by a much greater degree of warmth and of dryness during the winter season than might be expected from its geographical position. The winter temperature is, roughly, about ten degrees higher on the Riviera than on the south coast of England.

The Riviera is suitable for a very large class of invalids who have not vigour enough to respond to the cold of the Alps. Old people especially, with tendency to bronchial catarrh, do well.

Egypt, though warmer and drier than the Riviera, may practically be considered suitable for the same class of cases.

The winter climate of the Canary Islands resembles in many respects that

Digitized by Google

of the Riviera ; but it is not so dry. It is suitable for the same class of cases —cases of not very active disease in persons of somewhat low vitality.

Madeira has a much less dry climate than any of the places mentioned. Though it has lost its former popularity, it is still useful for many patients of a low grade of vitality, and especially for persons with renal complications.

The English south coast affords the most suitable winter quarters for many invalids. Bournemouth and Ventnor have deservedly been favourites for many years. Cases with serious digestive troubles and with tendency to diarrhœa do better there as a rule than at the Continental resorts. Persons in fairly good general health, with chronic disease not in a very active condition, not infrequently get on better on the south coast of England than elsewhere.

A word or two may be said touching leading symptoms in regard to the choice of a resort.

A high degree of fever, 102° F. in the afternoon, generally means that for the time being absolute rest is indicated; and a journey, especially to a foreign or distant health resort, ought as a rule to be postponed till a less active condition is reached. A normal or subnormal temperature in the morning and a temperature of about 102° F. in the afternoon is not necessarily a contra-indication either to travelling or to the selection of an Alpine or Mediterranean resort. But in such a case the medical adviser should be very sure of his ground before he counsels such a step. A temperature over the normal in the morning, say 99° or 100° F., and reaching 101° or 102° F. in the afternoon, is usually best treated by rest at home rather than by change of climate.

In regard to hæmoptysis a distinction has to be drawn between the hæmorrhages of early phthisis (usually small) and the hæmorrhages from large cavities in the advanced disease (usually copious). The early hæmorrhages get on well in any climate that suits the patient otherwise, hence the hæmorrhage need not be specially considered in making a selection. Cases of this kind with only slightly marked physical signs, as a rule, do remarkably well in the mountains. Hæmorrhages from large cavities, on the contrary, usually show a greater tendency to recurrence at a high altitude than at a low level. The seaside is popularly regarded as predisposing to hæmorrhages, but the tendency is probably an individual one.

A patient affected with tubercular disease of the intestines or with a marked tendency to diarrhœa will in most cases do wisely to remain in England; Madeira and the Canary Islands are especially to be avoided.

Irritable and inflammatory conditions of the pharynx and larynx are apt to be made worse by an excessively dry atmosphere. Such cases, for example, are apt to suffer much more discomfort at Davos than at Bournemouth. Tubercular ulceration of the larynx, however, without much surrounding inflammation or irritation—and this is a frequent condition—does well in Davos, provided the case is otherwise suitable. So much depends on local treatment and absolute rest for the voice in cases of tubercular laryngitis that patients should be under the close observation of a

Digitized by Google

competent medical man, and as a rule in a sanatorium, where the loquacity characteristic of these patients can be kept strictly in check.

Anæmia, though a consideration to be borne in mind in the selection of climate, is of only subordinate importance unless it be extremely pronounced in degree. The slightest degrees of anæmia, especially in young subjects, are usually much benefited in the mountains. Persons with extreme degrees of anæmia, on the contrary, have difficulty in accommodating themselves to the smaller supply of oxygen, and may easily suffer from heart failure.

I should like to lay stress on one point. Disinfection of rooms that have been occupied by tubercular patients should be insisted on at every health resort, and medical men sending out cases would do well to satisfy themselves as to the regulations in use where the patient goes.

In conclusion, let it be said that while some margin must be allowed for error and uncertainty, attention to the principles laid down will generally enable one to make a wise choice.

SIR J. W. MOORE, *Professor of Medicine R.C.S.I., Dublin.*

Sir John Moore considered that climate played but a secondary part in either the prevention or the cure of consumption. We should not forget that the vast majority of tubercular patients had not the means or the opportunity of changing the climate in which they lived. They contracted the disease in overcrowded tenements, and were too often profoundly ignorant of the danger of infection by the sputum. The speaker instanced a case in which a coachman infected his wife and son, who, after months of unceasing struggle, at last were enabled to resist the disease. Fresh air and sunshine were of the first importance in the treatment of the malady. Dr. Stokes told of a gentleman, apparently hopelessly ill of consumption, who went home to die, but indulged in duck-shooting, which open-air sport happily led to his recovery. Long sea voyages were of very doubtful advantage. The vapour-laden sea air, both on board ship and at coast health resorts, sometimes depressed those who were invalids, and this rather than the strength of the wind led to unsatisfactory results in the application of the treatment by sea voyages and residence at marine health resorts.

PROFESSOR CHRISTIAN GRAM, *Copenhagen*,

cited the experience of twenty-five years at the seaside hospital of Refsnäs (Zealand) in illustration of the successful treatment of surgical tuberculosis at the seaside. He felt doubtful as to the investigations which had been quoted with regard to the Isle of Man, as in Denmark they had found that tuberculosis was more abundant in the central portion of islands than on the shore. He believed that most—probably all—cases of pulmonary tuberculosis could be treated successfully at the seaside. It was always possible to minimise the influence of wind.

Digitized by Google

In Denmark they had consequently had all sanatoria built at the seaside. As it seemed true that tuberculosis could be cured in every climate, it was best that patients should be treated at the place where they must continue to live after treatment.

DR. F. M. SANDWITH, *Cairo.*

Every man who lives in a health resort becomes very early inoculated with the microbe—I do not know it by the Latin name, but it means the microbe of the universal belief in the place in which he lives. I have nothing to say about the advantages of Egypt, but there are one or two points I should like to mention to you, although you may know them. Those who have decided to send their patients out of England, rightly or wrongly, have, of course, the choice before them of the ocean voyages which we all know of, the advantages of mountain climates, which are very, very great, and the possibility of desert climates. In all the records which I have seen, and in papers written on the subject, the different advantages of these three are very fairly exposed, such as papers by Dr. Theodore Williams. He has always spoken of Egypt as a low-lying country—and it is quite true. Yet there is the interesting fact that if you take visitors, as I hope to take some of you some day, into the desert, you will at once be surprised by meeting air which reminds you of the kind of air you get at the top of a mountain in Europe—the ozone in the desert which you do not have in the towns of Egypt—which reminds you of the delight you have in climbing a mountain; yet Egypt suffers, as it were, from the stigma of being a low-lying climate. After the eighteen years I have lived in Egypt, I have only as yet seen one case of a European living in the country becoming tubercular there. I have, of course, seen a large number of people arriving with tubercle. This one man, who had been an officer in the Army, whom I met two months ago, had been in the Soudan. He came over to this country and to Europe generally, and I cannot tell you how he got the tubercle, but he believes himself that it was a relic of the typhoid fever which he got in the Soudan. That only one European resident should have in my experience contracted tubercle while living in the country is interesting. On the other hand, there is another interesting fact, viz., that the natives around us, breathing more or less the same air, do get tubercle; that, therefore, is an argument against climate in favour of the general sanitation and hygiene. The native does not get the same food, and he does not live in sanitary houses, and he does not spend his time in the sunshine out of doors, but he remains cooped up in climates even worse than this room at the present moment. Besides the Europeans who do not get tubercle, with the exception of my one friend, and the natives who do get tubercle, there are a large number of people who are working in Egypt who came originally to Egypt because they were sent there for their lungs. That is an interesting class of case, of professional men and others who came to Egypt—I do not mean to die—they came there because they were useless for work in Europe, and they are able now to make considerable incomes because they are living in a fairly decent climate.

Digitized by Google

DR. AMREIN, *Arosa, Switzerland*, gave observations. *See* paper, page 57.

Paper by the late DR. CARL RUEDI, *Arosa*, was read, specially prepared for this Section, *see* page 74.

MR. QUARRIER, *Glasgow.*

It is more than thirty-seven years since I first became acquainted with consumption. I got acquainted with it in working amongst the poor of the city of Glasgow. I found that the children of the poor were infected by their parents and the localities in which they lived, and how to get a process of cure was a difficult problem in such surroundings as we possess in the city of Glasgow, given up almost wholly to manufacturing. In other words, the atmosphere of Glasgow is not a place where consumptives can well get through with their trouble. Twenty years ago I was led to rent a farm in Renfrewshire for the treatment of children generally, and particularly children that were troubled with consumptive tendencies, and I found that the pure air of Renfrewshire had a wonderful influence on the young life of those children that were born of consumptive parents. It occurred to me that in transferring numbers of these children in Scotland to Canada, where there is a different atmosphere from what we possess in Scotland, there would be good results, and the effect of twenty-seven years' trial there in taking out children of consumptive parents is that I have found large numbers of those that were infected by their parents have lived to an unlimited age; whereas had they been left in Glasgow, or in Scotland, they would have been dead long ago. Therefore that is something to know about the climate or the air.

By-and-bye we were led to look at the treatment of adults as well as children, and some ten years ago we bought another farm and commenced to work amongst the consumptives of Scotland—that is, the consumptives that were not able to travel to the Continent, consumptives that were not able to pay for sanatoria, consumptives that were not able even to earn their daily bread—at least some of them; and how to make provision for such an army as these was a very difficult question. Well, notwithstanding the work of the orphan homes of Scotland, of which I am the originator, I looked to God for the money to build the sanatoria, and we got £10,000 to build the first, and we have £12,000 to build the second, and also £10,000 to build an Executive Work House, a house including a mortuary and a laboratory, and including all that is possible to be carried out by medical science, as the result of a few years' treatment. There are two houses, holding eighty patients, for females, and we are looking forward to getting money to build houses for males, for we find that the disease is equally great amongst the working-class males as amongst the working-class females. We also find that out of about 300 patients that have been treated during the past few years, something like 22 per cent. have been cured. Then there have been about forty-

Digitized by Google

four cases that were comparatively cured, and the rest of the one hundred just like what we have heard of to-day. Now, what I want to bring out, Mr. Chairman and friends, is this, that climate has not so much influence as locality. The Riviera, and all those other places that have been named, have not so much to do with the cure of the disease as the practical treatment of the patient when you get him in hand, and I have been wondering why there has not been more attention paid to-day to such a climate as Scotland. People say, "It is always raining there, and certainly if it is not raining it is snowing, and if it is not snowing it is boiling," and therefore we have a climate in Scotland that is almost the worst in the world. That has been demonstrated by medical scientists in Glasgow. Dr. Duncan, of the Victoria Infirmary, one of my Advisory Board, if he had been here would have backed up my statements about the results in the most unsatisfactory climate in the world. You men that are living apart all over the world, take courage, and look well to what science has taught you, and what common-sense teaches you every day; you men, look and see that the patients that you deal with from time to time get a chance before they have gone beyond the reach of cure of anyone but God alone!

DR. E. HOBHOUSE, *Brighton.*

The nature of the disease does not enable one to determine the climate which is suitable for its treatment. The question, to a large extent, is what climate suits the individual constitution. Some patients cannot stand the cold of the high altitudes, others the enervating effects of such climates as Madeira, the Canaries, etc. For the most part, I can only endorse the remarks of Dr. Huggard—that the character of the disease does not indicate the climate most suitable for treatment. Cases with uncompensated heart disease, albuminuria, and those which have supervened on bronchitis and emphysema, on the whole, are unsuitable for high altitudes; but even here there are very wide exceptions. Very remarkable and unexpected results are seen even in patients whom one would never expect to be benefited—*e.g.*, in patients with old fibroid disease and labouring hearts.

I should like to say one word about sea voyages, because they are, in my opinion, extremely risky. There is no doubt that they are extremely beneficial in some cases, but, on the other hand, there is no question that, when the conditions are bad, they are so bad that nothing can well be worse, and those conditions are not such as can be altered by any human agency. Therefore, I say that great care ought to be exercised in recommending patients to take long sea voyages.

DR. CROSBY WALCH, *Tasmania.*

The climate which I wish to bring under your notice is that of a country at the other end of the world, comparatively little known to you. I refer to Tasmania, commonly known as the Sanatorium of Australia.

Digitized by Google

In Tasmania, though as yet we have no systematised sanatoria, we have during late years been carrying out the broad principles of open-air treatment, and the climate, although insular, is, judging by the results we get in the treatment of phthisis, very suitable for the purpose.

The island is particularly mountainous, and for the greater part covered with extensive eucalyptus forests; and it is in the mountains of the interior of the island, surrounded with the antiseptic eucalyptus trees, that I have seen many patients improve in a remarkable way, and in some a complete arrest of the disease.

Our climate resembles the best points of the English climate almost more than any other in the world. We get no extremes of heat and cold; in Hobart, the capital, the mean temperature is 54·5, the average number of rainy days in the year is 170, and the amount about 20 inches, and the atmosphere is very free from dust and remarkably clear.

The Government has been approached with regard to their helping to establish an open-air sanatorium, and they have undertaken to at least give the necessary land for the purpose. Such cases of phthisis as Dr. Burney Yeo calls the rheumatic fibroid do especially well on the warm, dry east coast of Tasmania.

Dr. EXCHAQUET, *Leysin.*

Sur les contre-indications de l'altitude. Les réactions personnelles sont très variables, mais en général les formes suivantes se trouvent peu bien de l'altitude et du froid.

1° Craint exagerie du froid, mal supporté par certaines natures.

2° Emphysème et lésions superficielles étendues, limitant trop le change de l'hématot.

3° Affaiblissement du cœur dans les cas chroniques et accélération habituelle des mouvements du cœur.

4° Cachexie cela va sans dire.

Dr. C. THEODORE WILLIAMS, *London*, in reply, said: A great many of the remarks and the criticisms have been well replied to by the other speakers. The discussion has gone over a great deal of ground and we have heard new facts of various countries, and we have also heard of a good many sorts of treatment. The only regret I have is that we did not keep quite enough to climates. I put tables before you with the view to having climate and climate only discussed, but there was a great deal of discussion on the treatment of consumption and sanatoria. However, I am very well satisfied. We have had some very interesting papers and discussion, and we have had an amount of experience from the different speakers which I suppose is almost unequalled in variety.

In this discussion my dear friend Sir Hermann Weber took exception to my views about wind. I am aware of what he said about the south-west wind

Digitized by Google

in England, and I am quite aware that in certain localities (which papers read in these rooms showed) there was a greater prevalence of phthisis mortality in parts of Devonshire, where there is no protection from the south-west wind, than in parts where it is protected. But really it is the damp. The people who know the south-west wind in England, particularly in Cornwall and Devonshire, will know that is one of the most searching winds going. For instance, the moisture it carries with it will go right through a stone or a brick wall, and then people have all they can do to keep it out. They keep it out by ingenious practices—tarring, slating, and putting the slates in a particular way; but of course that only applies to larger private houses. There are a great many houses, and particularly in some parts of Devonshire, which are built with earthy clay, and there no doubt the south-west wind would penetrate right through. There you get damp walls. Damp walls would go a long way towards predisposing to consumption. That, I think, is the explanation of a good deal of what Sir Hermann Weber has said. I do not agree about the wind, but I am an Englishman and, like most Englishmen, I am accustomed to it. He is a German, and he does not like it. You will find that is so in a variety of ways when travelling abroad, while the English race and the Irish race do not object to it. I admit there must be a certain amount of shelter for our invalids. With regard to sea voyages, I tried to guard myself in every possible way. I said, "Providing that the food was good, providing they had cabins so that they got free ventilation," and I did not say anything about sea-sickness, because I hope we all get over sea-sickness after a while. But if anybody is incurable with sea-sickness, then he had better not go to sea. If people go to sea they must not expect to be coddled up; you expect patients to be out in all weathers on land, so you do at sea; you expect them to put on their tarpaulin, and walk up and down the deck and not mind any kind of weather at all. You must remember that is necessary for sea voyages. You must not put the patient on the same principles as a very weakly creature in a sanatorium; he must be ready to stand any weather, and then he will be able to reap the benefit. A certain number of cases have improved very greatly.

Another point was raised by Dr. Sandwith, who I was glad to hear make such a very sensible speech about the climate of Egypt. Mr. Quarrier, of Glasgow, spoke of the advantages of the orphan homes in Scotland and what great advantages the children get. There is no doubt about that. I do not consider that phthisis cannot be treated in this climate; it can be treated, but we have to find out in what climate it can be most successfully treated. There are a lot of cases that do not do well in England, and if we transfer them to some of those places I have been speaking of they turn out magnificently. Those are the cases I have to deal with. So long as they manage in England well, then do not send them away. But every autumn we have to set the plans for a large number of folks.

I have tried to show some of the conclusions I have come to, based on the experience of some hundreds—I might almost say thousands—of cases, and these are the conclusions I have come to, and certainly as regards the

Digitized by Google

ordinary tubercular cases—tuberculisation—if they can take exercise and so forth they will do well in this country; but they will do better in high altitudes. As regards the rest, I think we are all agreed on the plan proposed, that we all want to give our patients as much good air and good food and exercise as they can stand, and we have to adapt our climate to them. As to putting aside climate altogether, because you have to consider how you can carry out the sanatoria treatment in any climate, I cannot understand it; in a good climate you have, in addition to sanatoria, the climate. so that you have another string to your bow.

A QUERY AS TO WHETHER ANY SPECIAL TYPES OF PULMONARY TUBERCULOSIS AND ITS COMPLICATIONS ARE UNSUITABLE FOR TREATMENT IN THE CLIMATE OF THE BRITISH ISLES.

By P. S. HICHENS, M.B., M.R.C.P. (Lond.).

Dr. Hichens remarked that we have heard very much hitherto about the successes of the sanatorium treatment, but very little about its failures. According to some writers, one would imagine that there were no failures, but yet, even excluding the most advanced cases, and cases in which acute miliary generalisation and violent hæmorrhages intervened, there were undoubtedly cases whose rate of progress was at any rate a considerable disappointment to one. Were these disappointing cases of any special type of the disease or its complications?

So far as his own small experience had gone, he had had two types of disease whose rate of progress had been a disappointment to him. In one type the disease had assumed *a bronchitic form*, in the other the disease of the lungs, in whatever form, was associated with persistent troublesome diarrhœa with very irregular pyrexia. Two cases illustrating these types of disease were described. These cases had improved but very slowly, and not nearly so rapidly as other forms of disease, which might at first sight have appeared much more serious. With the exception of acute pneumonic cases, and cases associated with pleuritic effusion, he had had most of the ordinary forms of tubercular disease of the lungs and its complications under his care. The ordinary rapidly-spreading broncho-pneumonic forms, chronic forms with excavation, and associated with severe tubercular laryngitis and tubercular peritonitis, had all done extremely well. He thought it would be of great interest to know whether others had had the same experience in other types of disease in whose progress they had been disappointed. Would these types do better in a more perfect climate, or would their progress be disappointing under any circumstances?

The excellent results obtained by the sanatorium treatment of consumption in the British Isles were now known to all, and he thought our knowledge would now be better advanced by discussing our failures than by boasting about our successes.

Digitized by Google

ON NATURAL IMMUNITY FROM TUBERCULOSIS IN NATAL, SOUTH AFRICA.

By JAMES F. ALLEN, M.D., *Delegate from Pietermaritzburg, Natal, South Africa.*

Situation, Topography, and Climate of Natal.
Natural Immunity from Tuberculosis among Cattle, Monkeys, Europeans, Zulus, Indians.
Visitors suffering from Pulmonary Tuberculosis.

In considering the means whereby the disease known as "Tuberculosis" may be repressed, climatology must always take an important place; it being a matter of common notoriety that in certain parts of the world, under certain conditions of climate, topography, and social relations, this disease is a scourge which almost threatens the existence of the human race; and again in other regions, and under different circumstances, it seems to have lost its power of injuriously affecting man, though in some degree it may be known to exist locally. How the human race can exist in any country where this disease is known also to be, and be practically free from its effects, is a question so interesting, important, and instructive, that every effort should be made to explain such immunity. For what is a natural mode of defence in one country may be artificially produced in another, or at least so nearly approached as to give great protection, and by the knowledge thus obtained those predisposing causes, and circumstances which favour its propagation, can be lessened or removed.

The evidence, therefore, of anyone who has practised the medical profession, and who has lived for many years in a land so favoured, with reference to this disease, that Nature has made it practically innocuous there, and who has at the same time had considerable clinical experience, not only in general practice but in the charge of a public hospital, should be of some service in this enquiry.

I have lived in Pietermaritzburg, the capital of Natal, for twenty-six years, and during the whole of that time I have been in charge of its public hospital, known as Grey's Hospital. I have also been the greater part of that time Medical Officer to the Corporation, and have thus been brought into intimate relations with all sections of its inhabitants, and have had its local conditions, socially and physically, constantly under consideration with reference to public health.

Pietermaritzburg is situated in the middle of the Colony, about forty miles from the sea, in a direct line, at an altitude of 2,200 feet. It is built at the foot of the southern spur enclosing the Zwaart Kop Valley, and is therefore in a very dry and sheltered situation. Its population numbers 30,000 people, of whom about half are coloured, and consists of natives—that is Zulu Kafirs—Indians, and people of mixed race; the other half being Europeans.

Digitized by Google

This city has nominated me its delegate to this Congress. Having thus presented my credentials, I will proceed to describe the climate and topography of Natal, and attempt to show how local conditions have modified the severity of Tuberculosis in that Colony.

Natal is a part of South-eastern Africa, somewhat diamond-shaped, having a coast-line of about 200 miles, north of St. John's River, and an area of about 20,851 square miles. It has a mixed population, being made up of 60,000 Europeans, 1,000,000 Zulus, and 50,000 Indians. Three such races, wholly dissimilar in habits, origin, and religion, present a diversified field for observation with reference to the prevalence of any disease, and cannot but be instructive with regard to tuberculosis, from which deductions possibly can be made as to what locally tends to check the disease, and what common boon these totally different races enjoy which appears to give each an immunity from its attack.

Natal is the last portion of healthy South Africa east of the Drakensberg range. The healthiness of the eastern coast of Africa, from Capetown to where Natal ends, depends upon the situation of the Drakensberg Mountains. This range begins at Table Mountain behind Capetown, and as a guardian to human health ends at St. Lucia Bay in Zululand. After it passes St. Lucia Bay, the range extends far north into the continent of Africa, but from St. Lucia north it ceases to be a factor in the preservation of health, as far as human beings are concerned. Indeed it becomes a potent means of rendering the country almost uninhabitable to Europeans—thus forming a vast region where wild and savage beasts may be preserved, to await their own epoch for disappearance. Be that as it may, the Drakensberg range from Capetown to St. Lucia Bay secures a good climate to that portion of the African continent, and defends it from malaria. But north of St. Lucia Bay, it causes such a condition of soil saturation as to form a developing ground for the parasite of malaria, of such vast extent as to render hopeless all human means of making it healthy.

That the same mountain range should be the guardian of health in one region, and in another be the cause of the country being almost uninhabitable, needs some explanation; and as it will help to throw more light on the physical features and condition of the healthy district which I am considering, some further reference must be made to it. The Drakensberg range, from Table Mountain at Capetown, runs north-east, following the coast-line to St. Lucia Bay, for a distance of nearly 1,000 miles, the last 200 of which forms Natal. At St. Lucia Bay the mountains trend to the west, leaving the coast and gradually drawing inland, having an ever-increasing plain between the foot of the mountains and the sea. The result of this configuration is, roughly, to divide the eastern littoral of Africa into healthy non-malarial and unhealthy malarial districts, St. Lucia Bay being the dividing point.

During the spring and summer, on the eastern coast of Africa from Port Elizabeth northwards, the south-west wind blows on to the land, laden with moisture from the ocean, and it is from this source that Natal receives its plentiful supply of rain from October to May, which cools and fertilises the

Digitized by Google

country, keeps it healthy, and renders it a suitable place for European settlement. On the other hand, it is also this rain-laden wind which, north of St. Lucia Bay, makes Africa, by the operation of the mountains, practically uninhabitable from malaria. The foot of the Drakensberg, from Table Mountain at Capetown to the lagoon known as St. Lucia Bay, is practically conterminous with the sea. On this account, moisture condensing into rain and falling on the range, runs directly into the sea, there being no flat land intervening to receive it. Natal being part of the side of that range, its rainfall descends on the eastern watershed of the Drakensberg, and runs directly into the Indian Ocean, leaving the earth and air dry and cool. But from St. Lucia Bay north-east of Natal, on account of the westward trend of the mountains, the rain which falls on the Drakensberg, increasing from occasional thunderstorms in Natal to a torrential downpour as the mountains pass north and approach nearer to the Equator, does not flow directly into the sea, but is in a great part absorbed by the ever-increasing plain which, as the mountain foot recedes westward, interposes between the range and the sea. From this vast plain arises the malaria which makes Eastern Africa uninhabitable for Europeans. The non-saturation of the soil of Natal is not only demonstrated by these physical features, but also by the fact that it is completely free from malaria, which it would certainly not be if it were in the least water-logged. It is necessary thus to fully explain the origin of the malaria north-east of Natal, to make it clear that the soil of that colony is dry, and its surface and subsoil well drained.

Anyone watching a rainstorm forming in Natal would be surprised to notice that the clouds seem to come from the interior, where nothing but the waterless continent of Africa exists, the apparent marvel being that rain seldom or never seems to come from the direction of the sea. Great masses of cloud can be seen forming on the Drakensberg, or rapidly condensing and passing down the spurs of that range to the ocean, the moisture from which the rain is formed, having passed eastward with the south-west wind until stopped and condensed by the mountain range in the interior, often with the grand electric disturbance so well known as the Natal thunderstorms.

The Drakensberg range runs north-east through Natal, following the coast-line, its spurs south-east, practically, if irregularly, at right angles to the main range. Rivers, having their origin in the highlands, run down its valleys in an easterly direction to the sea, rapid mountain streams for the greater part of their course. The prevailing south-west rain-producing wind of the summer is occasionally interrupted by gusts of hot, dry wind from the north-east. The latter, being evidently obstructed currents, blow directly on the earth, thus having the power of raising the dust, and are the cause of the dust storms of Natal, which are the most trying feature in the climate. But, fortunately, they are not of frequent occurrence, being most prevalent in August, September, and occasionally later in the summer, but occur on the whole not oftener than a dozen times in the year. Apart from the power they have of raising the dust, which away from the main roads and towns is very limited, they are not unhealthy. The atmospheric obstruction is of short duration, the hot wind

Digitized by Google

seldom lasting more than a day, and often only a few hours, and is always followed by the cool, rain-carrying south-west current, which, being parallel with the earth's surface, does not raise the dust, and being moisture-laden soon condenses into rain, refreshing and cooling the earth. Thus it will be seen that the prevalent winds in Natal blow across the valleys, not up or down them; the result being that in almost every valley there is an exposed and sheltered side, and a point of greatest rest; the exposed being the south-west face on the north side of the valley, on which beat directly the wind and rain from the south-west, and is protected from the dry, hot north-east winds. On the other hand, the south side of the valley, with its north-east aspect, is dry and sheltered from the rain-storms and is open to the hot wind. The result on the vegetation of these valleys is remarkable; the bush almost invariably occupies the south-west aspect of the upper ridges of the northern slopes, and the grass is rank and sour, all directly the result of exposure to the south-west rains; and being protected from the north-east hot winds, the ground retains moisture throughout the year in a sufficient degree to enable both grass and bush too, not only to grow, but to resist the effects of the grass fires. So marked is this arrangement of the vegetation, that the bush stops in a sharp and straight line along the tops of the hills, as if it had been so planted by the hand of man, and the grass becomes shorter and sweeter, the exposure to rain being less, and the drying greater. Deep in the valleys on the south side, well open to the north-east, so as to escape the heat of summer, and high enough from water to avoid the cold in winter which is always associated with water, is the best situation for a residence. Such is the situation in which Pietermaritzburg is built, and the choice of the site shows the knowledge of the country possessed by its Dutch founders. This rough landscape and weather guide applies more particularly to the highland and midland districts of Natal. Those seeking health there, suffering from or threatened with pulmonary tuberculosis, and who may wish to combine farming with that pursuit, must never forget that shelter is the great desideratum for man and beast in South Africa.

Natal, being practically the side of a mountain, possesses three fairly distinct climates, and has, as a result, a very wide range of products. That of the coast, a warm and semi-tropical district; humid during the spring and summer, from September to May, on account of its proximity to the sea, and the prevalence of the sea breeze during that season of the year. The average mean maximum temperature throughout the year in this region is 77·8°. During the autumn and winter, from April to October, the atmosphere is bright, dry, and free from frost. In this district tropical fruits grow well—oranges and other citrus fruits, mangos, pineapples, bananas, guavas, and such productions as sugar, tea and coffee, maize (known locally as mealies), millet (or Kafir corn), and tobacco. It is not a good cattle country, but cattle can be bred there; sheep do not thrive; poultry do well; and it is thickly populated, for South Africa, by natives and Indians. But it is not a region in which Europeans flourish.

In the midlands the climate, at an altitude of from 2,000 to 3,000 feet, is

Digitized by Google

very healthy and suitable for European settlement. The rainfall is about 30 inches per annum, and the mean temperature is considerably lower than the coast, the atmosphere is much drier, and there is a distinct fall in the temperature at night, which is refreshing, invigorating, and induces sound sleep, the absence of which is perhaps the most debilitating characteristic of the coast, and during the winter frost at night is of common occurrence. The ground from the sea rises rapidly, ridge after ridge, into apparently confused hills and valleys ; until within 40 miles of the sea, the altitude of the whole country has risen to over 2,000 feet, the landscape is open and undulating, well watered, and almost free from bush. Horses, cattle, and sheep thrive; mealies, millet, potatoes, oat forage, green barley, peas, beans, and tobacco grow well; citrus fruits of all kinds, apples, pears, peaches, and plums flourish. It is the home of many European farmers, but it is by no means fully occupied, and would support a much larger population than it carries at present, and there can be no doubt but that, when South Africa is thoroughly known, it will do so. Within it are high and at the same time sheltered districts, peculiarly suited to persons threatened with or suffering from tuberculosis; where in the case of actual disease recovery may take place, or strength may be maintained by continued residence, and where feebleness of constitution without disease exists, security from invasion may be secured. The highland, up to the foot of the peaks of the Drakensberg, from 3,000 to 6,000 feet above the sea, is an open, rolling, grassy country, covered with sweet, short herbage, on which stock of all kinds thrive. Indeed it is the stock country of Natal; for horses, cattle, and sheep there is no better district in South Africa. The atmosphere in summer is cool and dry; cold and frosty in the winter. During the spring and summer, from September to May, thunderstorms and hail are not uncommon, and are often destructive to fruit and crops. But during the winter, from May to September, no rain falls. The district, however, is so well supplied with running streams that nothing but the grass suffers from drought. Then, indeed, the country is dry and the grass parched, but for all that, cattle reared in the district can find subsistence until the spring grass appears, which is always early. But as the breed of horses and cattle has improved, and lands become enclosed, farmers have been compelled to provide some winter food. Grass cut on the veldt and made into hay, with very little else, is found to be sufficient. This district will, in the future, be not only the principal dairying country, but it will produce sufficient meat to supply the whole colony, for there the largest and heaviest breeds of cattle can be reared.

European fruits of all kinds thrive. There are to be found the most successful orchards in the Colony; mealies, wheat, millet, oats, barley, turnips, peas, beans, and potatoes grow well, and human health is excellent. Thus it will be seen that the climate in Natal is very dependent upon altitude, but though it is thus varied, in the main it consists of two seasons—the summer, in which there is rain, and the winter, in which there is none. During the summer the heat is often trying, but in the midlands and highlands is greatly relieved by the fall in the temperature at night. During the cold

Digitized by Google

months, from the end of April to September, the whole atmosphere of the country, from the sea to the summit of the Drakensberg, is cool, dry, and still.

But no description of the climate can adequately convey to the mind the exhilaration of spirits and body produced by the certainty, almost, of pleasant weather and unfailing sunshine, with the possibilities it affords of open-air life, and which the inhabitants lead.

If we search for tubercle in Natal we will get some instructive information if, in the first instance, we seek for it among cattle born and reared on the veldt of the Colony. The cattle districts of Natal are the midland and highland terraces of the Drakensberg, especially the latter, at an altitude of from 2,000 to 6,000 feet above the level of the sea. Though the highlands are the best cattle-breeding districts, and the largest and finest breeds can be reared there, being comparatively free from ticks, red water, and other pests which retard the success of cattle-farming elsewhere, as yet it is in the midlands that the greatest number of cattle are to be found, the highlands being but sparsely inhabited either by natives or Europeans; the midlands thickly populated by natives, who choose these sheltered districts on account of the mild climate, where they can grow mealies, millet and tobacco in profusion, and there they will not be exposed to sharp cold in winter, as they would be in the higher altitudes. Here also their cattle get through the winter better, their owners not being put to the necessity of providing special food and shelter for them, as cattle there can, under normal conditions, all the year round, find sufficient food on the veldt and shelter from the wind. The natives must have been well aware of the superior character of the country under the ridge of the Drakensberg for maintaining flocks and herds, if from nothing else, by the fact that during the spring and summer, before Europeans had settled in this district, the veldt was covered with game—antelopes of many different kinds in thousands, and great herds of quagga; but all its recommendations sank to nothing in comparison to the inducements offered by the midlands, and to the fear that the great Zulu possessed for the little Bushman with his bow and poisoned arrows. For in former times, before the Bushmen were exterminated by Europeans, this weird race of savage mountaineers dominated this region and drove the Zulus from the sweet grass of the highlands. The last spurs of the Drakensberg were the home of the Bushmen; here in the caves and rocks they could lurk in safety, and from these heights could survey the whole country spread out before them, and they had to hand the latest news of importance, as far as they were concerned, in those strange but life-like figures and drawings on the walls of caves and the protected faces of cliffs, whereby one roving band could convey to another the dangers and benefits to be found in each locality. Cattle thieves by nature, the country offered them every facility for indulging the habit. Stolen cattle once in the defiles of the mountains were lost to their owners, and if overtaken before they reached that refuge, with their poisoned arrows the Bushmen were always able to make a good defence against their pursuers. No wonder the cattle-loving Zulu gave them a wide berth, and even looked

Digitized by Google

upon them with superstitious fear. This is the simple explanation of the uninhabited condition of the highlands of Natal on the advent of Europeans, and clears that part of the country from the least suspicion of unhealthiness. The midlands was, and is, the home of the Zulu. Here he possessed, before the rinderpest swept over the country, perhaps a million head of cattle—small, hardy Zulus, suitable to the country. These animals, and, indeed, those belonging to Europeans, may with truth be described as wild cattle. A very few were tame enough to submit to being milked, but otherwise they were as free as the buck. They lived in a natural state, unaffected in any way by artificial surroundings, and whatever their state of health might be, it would be the reflex of the local natural condition of the country in which they were reared. If that land had a natural tendency to develop any disease which would affect cattle, then undoubtedly these cattle must suffer from it, as nothing was done by man to protect them; the country, too, being practically unfenced, they were fully exposed to contagion. Thus it was that rinderpest, in a few weeks, swept through the land, and for the same reason lung-sickness (pleuro-pneumonia) devastates whole districts. Therefore the condition of the veldt cattle, with reference to tubercle, should be an indisputable piece of evidence as to the prevalence of that disease in the country. As the greatest number of cattle are to be found in the midlands, which experience suggests is not so likely to be as free from tuberculosis as the highlands, the field of enquiry cannot be said to be selected for the purpose of coming to a favourable conclusion. The result of observation and experiment, as far as it has gone in Natal, is to prove that the wild cattle—that is, the locally bred cattle running on the veldt—are free from tubercle. Though such an immunity goes far to prove that the disease is innocuous to animals living in a natural state on the veldt, yet it does not prove that tubercle does not exist in the Colony. That it does exist there is easily demonstrated by examining dairy cattle. If healthy wild cattle are caught, handled, confined in cowhouses, fed, milked, used and treated as dairy cattle, they seem to lose their immunity against tubercle, may become affected with it and suffer from it in the same degree as dairy cattle in England. The immunity possessed by wild cattle in Natal is, therefore, not directly due to climate. This is an important point to establish for those who have to contend against the disease in climates which are supposed to be favourable to its development.

Again, take the well-known history of another animal, the monkey, with reference to this disease. These animals, captured in a healthy condition, carried to Europe, and confined in cages, even though the greatest care is taken that their surroundings are healthy, and the air they breathe is warmed, yet they die in great numbers from tuberculosis. Neither the cow nor the monkey eat meat or drink milk, and could not be affected from their food, yet neither a warm climate nor food free from tubercle was sufficient to protect them. Both fell victims to the disease on losing immunity, and both in common suffered this loss by being subjected to a complete change in environment and to confinement. There being ample evidence to prove

Digitized by Google

that tubercle bacilli are to be found everywhere, the tissues of these animals must have been able to resist the growth of the bacillus in their wild state. Contact is, therefore, not the cause of the invasion—that must have taken place frequently before. The change is in the animal itself; it has become vulnerable. Exactly the same set of events occur with reference to the bacillus of putrefaction. When death takes place, immunity from its invasion disappears; it develops in the body, and decomposition follows, exposure to the bacillus being the same before as after death. Something has died in the cow and monkey on their confinement, which permits of the bacillus of tubercle developing. What, then, is this natural immunity which is thus capable of resisting the growth of a bacillus under one set of circumstances, but is lost under another? It is evidently the result of bodily perfection, and is the product of selection. It is very different from scientific immunity, though its results, while it continues, are the same. It does not last as long as that caused by vaccination against small-pox; but natural immunity, being dependent on selection and perfection, is more to be desired than scientific, which has nothing to do with either, and can be established in the weak and diseased, while that of nature can only come after the weak have been made strong and the diseased are cured, and is thus always salutary, whether it is applied to the individual or to a race or species. If we can discover Nature's secret and adopt it, or as much of it as climate and circumstances will permit, we will be advancing undoubtedly on the right road. We may thus, in a great measure, supersede the necessity for the discovery of scientific methods for establishing individual immunity, which, though extremely important from a personal point of view, are essentially destructive, as they tend to preserve the unfit with the fit, and which, with reference to this special disease (tuberculosis), may not be attainable.

That such natural immunity does exist, to a greater or lesser extent, against the invasion of almost every specific bacillus, no one with any clinical experience will deny. Apparently such immunity differs with the bacillus, one set of circumstances making the body liable to the invasion of that of tubercle, another to that of tetanus, and a third to that of putrefaction—and so on. But these three bacilli, in relation to the human body, have much in common, and apparently demonstrate this theory of natural immunity very clearly. The bacilli of all three seem to exist everywhere and in any place. A healthy living body can be made immune against their natural invasion, while, on the other hand, means can be found to cause it, in the ordinary manner, to become affected by all three. The law seems to be, that when immunity is lost invasion follows. The futility, then, of fighting such bacilli becomes clear. As a side-issue in the campaign it is right and desirable, but as a central idea it is wrong and misleading. For instance, inspecting food for tubercle is not of much use if the consumer is permitted to live in an abnormal environment under such circumstances as tend to cause loss of natural immunity. Invasion and growth of the bacillus must follow, whether it enters by food or otherwise. The absence of tubercle from the food of the cow and the monkey does not protect them, and the

Digitized by Google

absence of tubercle from the food of human beings confined in a city slum will not save them. Attempting to destroy the bacillus is beginning at the wrong end of the problem. Greater attention should be paid to that which is attacked than to that which attacks.

Human beings in Natal are almost as free from tubercle as its cattle. Tuberculosis in one or other of its forms is to be met with, but the number of such cases is so small, and as the disease shows no signs of increasing, as a local product, its existence is more a warning than a danger. Tubercular disease of the glands, or of the membranes of children, is as rare as pulmonary disease in adults, and tubercle of the skin is very uncommon. When therefore 60,000 people are exempt from the well-known ravages of such a complaint, something must have developed in them a natural immunity which is capable of resisting the growth of the bacillus. The presence of the disease among the people, in the few cases which occur, shows that it is immunity, not freedom from exposure, which keeps the disease in subjection. What then are the conditions under which these people live which have developed this immunity? Undoubtedly the open-air life, which the benign climate and healthy country has made possible. As it formed and maintained this resistance in cattle and monkeys, it develops the same power in man.

The best Natal house is but a shelter from the sun and rain. It is as a rule only one storey, with matchwood ceilings and well-ventilated roof. The people sleep with the windows open at night. Each family has a house to itself; often a piece of garden. There is no overcrowding, no one is deprived of fresh air and light. The towns have no echoing streets of three and four storeyed houses, which exclude light and air from each other and the street between. There are no flats or slums. There is in Natal a tendency to abandon house life. The love of reading, essentially a house pastime, is not a characteristic of the country. Open-air games, riding, bicycling, and out-of-door occupations of all kinds are much sought after. In the towns business will be suspended by a cricket or football match of any note. All the shops must be shut, which only a very foolish trader would attempt to resist. In the cities, never since the Greek Republics has there been a community so given to gossiping on public affairs in the open air. When anything of interest stirs the public mind, the whole population turns out to discuss the matter in the streets. All the enquirer after public opinion has to do is to pass from group to group, and he will quickly know how the public leans. So well understood is this that members of the Government, well-known public men, newspaper editors, and politicians of all kinds can be met in the highways and byways gathering information. In no part of the world is the "man in the street" such a reality or so supreme.

So mild is the climate that people can sleep in the open air at night without risk. A favourite form of picnic is to camp out in tents for a week or a fortnight at a time; a blanket or less is considered sufficient shelter at night. There are no factories, and night work is almost unknown. It may not be possible to conduct the business of the world in such a manner, but those who so live are certainly more free from disease of all kinds, more

Digitized by Google

especially tubercle, than the hard-working, overcrowded populations of European cities.

The Zulu natives of Natal are a stalwart, healthy race, husbandmen by calling, and are in possession of a large proportion of the Colony, as theirs by right. They are a prosperous and happy people. The uncivilised Zulus can be said to be free from tuberculosis in all its forms, but with civilisation the disease appears. The Kafir follows the *rôle* of the cow and monkey in that respect. Confine him, alter his diet, make him wear clothes, the less robust soon lose their immunity, and fall victims to the disease. There is a high mortality among the children of the wild Kafirs, probably three times as great as among Europeans, but it is not from tuberculosis.

As with the Europeans, so among the uncivilised Kafirs, an occasional case of pulmonary tuberculosis is to be found. Tubercle of the skin is rarely seen.

The diet of these people in their wild state is quite different from Europeans. In Natal the latter eat more meat than would be considered wholesome in Europe, and is even less suitable to a climate like Natal. The Zulu Kafir lives in his natural state on maize, called locally mealies, made into porridge, or cooked in some other way, and curded milk, but the staple article of his food is Kafir beer or "tshwala," a thin fermented porridge, made from millet (Kafir corn). This Kafir beer leads to many a faction fight. Occasionally this diet is varied with a substantial meal of meat. "Tshwala" is prepared by allowing millet to sprout, thus malting it. It is then dried and stored for use, and at any time can be made into beer by a very simple process. It is first pounded into a rough meal, put into calabashes, into which is poured a sufficient quantity of water, and the whole allowed to ferment, during which time it is stirred, skimmed, and is kept at a temperature which aids the process. When it has acquired the right alcoholic strength, which is about that of "lager beer," it is fit for consumption. It is given to children a few weeks old, and does not disagree if sound and given with judgment. It is easily digested, as most of the starch in the grain has been changed by fermentation into glucose. It has a sour, pungent smell. Its taste is tart, pleasant, and agreeably refreshing. It is most sustaining, but is thickish and reddish-yellow in colour. It possesses the power of dilating the stomach after it is drunk by the quantity of carbonic acid gas it evolves—a most valuable property, where there is a tendency to contraction of that organ, as takes place in many wasting diseases, specially in phthisis, where contraction of the stomach, and consequent inability to take food, with failing appetite, tends greatly to hasten a fatal result. I have used it freely in wasting diseases of different kinds, and have always found it most invigorating. I believe it would be a valuable article of diet for the purpose of sustaining strength and combating waste in phthisis, not only on account of its digestibility, but also for its power of dilating the stomach. It contains a considerable quantity of alcohol, which is quite capable of causing intoxication, but this alcohol is of such a digestible character that it does not cause organic disease, nor does it demoralise, as results from drinking European spirits; on the contrary, it

Digitized by Google

helps to sustain and invigorate ; and certainly alcohol taken in that form, even though the quantity imbibed daily may be large, produces no predisposing tendency to tuberculosis. On the contrary, I think it would materially help in the curative treatment of that disease. The effect it has is to greatly increase the adipose tissue of the body. It is to the consumption of "tshwala" that the well-known obesity of Kafir chiefs is due. A photograph of Inkungu (The Mist), a brother of Cetywayo, the late King of the Zulus, shows this effect of Kafir beer.

In reviewing the vital condition of the Kafir race, some allowance must be made for the fact that every Zulu who arrives at adult age is a "survival of the fittest," the weaklings having been weeded out; but though that secures to a great extent a vigorous people, it does not prevent a failure of constitution under unfavourable conditions, with the loss of natural immunity against tubercle, which allows the bacillus to grow in the body. During their wild existence they live a life which tends to maintain what selection has started. They have no cities, consequently no overcrowding. Their grass huts are about six feet high inside—just enough space for a tall man to stand upright. These are not lasting structures, being often destroyed and renewed, change of site for his residence being a Zulu's commonest recourse for protection when serious illness has attacked his family ; and though he thus acts more from superstitious than hygienic motives, the result is alike beneficial. Thus their houses do not become the home of all the disease-producing bacilli which have generation after generation affected the residents, as is the case in the old, disease-sodden dwellings of the poor of Europe. To the Kafir, poor or rich, his hut is a friend, though it is but a shelter and a place to sleep in. To the poor of Europe his house is often a deadly enemy.

The lithe, wiry figure of the Indian, clad in his bright-coloured clothing, is a familiar and picturesque object in the Natal landscape. Fifty thousand of these people reside in the Colony. They have been imported chiefly for field labour, and are employed principally in the cultivation of sugar-cane on the coast; but though by far the greater number live there, they have spread far into the interior, and are to be found in every centre of population from Durban to Bulawayo. They belong to the labouring class in India, and are for the most part low-caste Hindus. Banyans—that is, Mahometan traders from Bombay—have also found their way to Natal, and many have acquired considerable wealth.

Residence in Natal causes the Hindu labourer to improve greatly in physique, especially noticeable in the second generation, the result of climate, country, and food: Natal being in all three respects for him a change for the better. They are industrious and healthy. Their houses are grass-covered huts or shanties, covered with corrugated iron or the tin of packing-cases. They are almost vegetarians in diet, living principally on rice, mealies, peas, fruit, dried fish, clarified butter, and occasionally a little flesh—mutton, goat, or fowl. They are by no means a temperate people, as far as the consumption of alcohol is concerned. They drink spirits freely, especially rum. They are practically free from tuberculosis in any of its forms. It is an exceptional

Digitized by Google

thing to find an adult suffering from pulmonary tuberculosis, nor do children suffer from it in any other region, and tubercle of the skin is very rare.

Here, then, in Natal, are two alien races, Europeans and Indians, differing in habits, customs, and food from one another, and from the native Zulus; but both, in common with the native, presenting the same aspect with reference to tuberculosis—almost entire immunity. The open-air life which each race lives seems to be the only common influence they enjoy which could account for it.

If the general hospitals in Natal are visited, cases of phthisis will indeed be found among the European patients, but almost invariably these are immigrants, mostly young men, and in the last stage of the disease. Latterly the number of such persons visiting the Colony in search of health has greatly increased, and has caused attention to be paid to the matter by the Government, so far that shipping companies have been asked not to give passages to persons in advanced consumption; otherwise no serious attempt has been made to stop the entry of such cases into Natal. But no country will long consent to such an invasion, and if it is not checked and some discretion exercised in the selection of cases, I feel sure the Colony will follow the example of New Zealand, and refuse entry to persons suffering from tuberculosis.

The great majority of the persons so suffering who have lately arrived in Natal are not only in a hopeless condition, but destitute. Very many come with the hope that, the country being a British Colony, they will be able to get employment; but as most are palpable victims of consumption, no one will employ them, and soon failing strength makes work impossible. They then pass through many stages of hardship, until finally they find their way to the hospitals, where they are permitted to remain till their death. But these institutions protest against receiving them, as their limited funds are thus used in the treatment of hopeless cases, which they should never have been called upon to admit. It is generally felt that the maintenance of such cases should be a charge on the country of their origin, which they should never have been permitted to leave.

If, in the future, Natal is to be used as a sanatorium for consumptives, destitute persons must be sent there under some recognised system, whereby they will be received and maintained in the Colony until they are well enough to support themselves, or until the end of their life. The present mode of casting them adrift is both inhuman and impolitic.

Digitized by Google

RÉSULTATS DE LA CURE D'ALTITUDE AU SANATORIUM D'HAUTEVILLE (AIN), FRANCE.

PAR MM. LES DRS. P. JONNART ET GUETSCHEL.

LES différents paragraphes porteront :

1° Sur l'état des malades à leur entrée (sélection des malades), état général, état pulmonaire.

2° État de ces mêmes malades au bout de 15 jours. Étudier les modifications physiologiques, pathologiques, et bactériologiques, survenues sous l'influence du changement de milieu et de régime.

3° État des mêmes malades au bout de 2 mois.

4° État des mêmes malades à leur sortie du sanatorium.

Statistique des résultats obtenus au sanatorium, après 9 mois de fonctionnement.

Conclusions : indications et contre-indications de la cure d'altitude.

RESULTS OF THE HIGH-ALTITUDE CURE AT THE SANATORIUM D'HAUTEVILLE (AIN), FRANCE.

BY DRS. P. JONNART AND GUETSCHEL.

THE different paragraphs refer :—

1. To the condition of the patients on their admission (selection of patients), general condition, pulmonary condition.

2. Condition of the same patients at the end of a fortnight. Sketches of the physiological, pathological, and bacteriological modifications brought about by the change of surroundings and *régime*.

3. Condition of the same patients at the end of two months.

4. Condition of the same patients on their discharge from the sanatorium.

Statistics of the results obtained at the sanatorium after nine months' working.

Conclusions : Indications and contra-indications of the high-altitude cure.

Digitized by Google

DE L'INFLUENCE COMPARÉE DES CLIMATS ET DE LA RÉSISTANCE INDIVIDUELLE DANS LA TUBERCULOSE EXPÉRIMENTALE.

PAR MM. LES DRS. LANNELONGUE, ACHARD, ET GALLIARD, *Paris.*

DANS des publications antérieures, nous avons fait connaître les recherches que nous avons instituées pour déterminer expérimentalement l'influence des climats sur l'évolution de la tuberculose inoculée au cobaye.* Le résultat de ces recherches fut assez imprévu : malgré les conditions en apparence le plus défavorables, ce sont les cobayes logés au laboratoire qui ont donné la mortalité la moins rapide, alors que les animaux placés à la campagne, au bord de la mer, ou dans la montagne, ont succombé plus vite.

Ainsi l'action du climat, dont la thérapeutique traditionnelle admet l'influence, sans préciser, il est vrai, comment elle s'exerce, ni même quel climat est le plus favorable, ne paraît nullement établie sur des bases expérimentales et vraiment scientifiques. Cette divergence entre les faits expérimentaux et le sentiment général des cliniciens commande l'attention. Elle fait, en tout cas, un singulier contraste avec la concordance parfaite qui règne, au contraire, entre l'observation clinique et l'expérimentation animale en ce qui concerne le rôle meurtrier de la fatigue musculaire, de l'alimentation restreinte, de l'inhalation de poussières ; toutes conditions qui précipitent de la façon la plus manifeste l'évolution de la tuberculose, aussi bien chez l'homme malade que chez l'animal inoculé.†

Les expériences que nous avons faites sur le rôle du climat sont intéressantes encore sous un autre rapport. Elles permettent de voir les variétés d'évolution qu'a présentées l'infection chez des animaux soumis à des conditions de nourriture et d'habitation identiques. Rappelons tout d'abord que ces recherches comprennent deux séries d'expériences ; dans chacune de celles-ci, 3 lots comprenant chacun 50 cobayes, inoculés en même temps dans le plèvre avec la même dose de virus, ont été placés dans des conditions climatériques différentes ; dans la première expérience, un lot demeure au laboratoire, un

---

* Lannelongue, Achard, et Galliard. "De l'influence du climat sur l'évolution de la tuberculose expérimentale pleuro-pulmonaire." 'Congrès de Naples contre la tuberculose,' avril 1900. 'Bulletin médical,' 2 mai 1900, No. 35, p. 406. 'C. R. de l'Académie des Sciences,' 21 janv. 1901.

† Lannelongue, Achard, et Galliard. "De l'influence de l'alimentation, de la température, du travail, et des poussières sur l'évolution de la tuberculose." 'C. R. de l'Académie des Sciences,' 6 mai 1901.

Digitized by Google

autre fut envoyé à la campagne, et le troisième au bord de la mer ; dans la seconde expérience, un lot fut également conservé au laboratoire, un autre fut envoyé à la campagne, et le troisième dans la montagne. Par suite, dans chacun des lots, 50 cobayes infectés de même étaient soumis aux même conditions de milieu. Or ils n'en ont pas moins présenté des différences considérables quant à l'évolution de la maladie.

Tandis que certains cobayes mouraient de tuberculose généralisée au bout de six semaines, au contraire, chez quelques autres du même lot, au bout de 15 et 18 mois, les lésions n'étaient que minimes et étaient restées localisées au thorax. C'est ainsi qu'à la fin de la première expérience, 3 cobayes du lot de la mer, 3 du lot de la campagne, et 4 du lot du laboratoire n'avaient que des lésions très discrètes. Et il en était de même, dans la deuxième expérience, pour un cobaye de la montagne et pour 3 du laboratoire.

La résistance remarquable que certains cobayes ont présentée à la généralisation de la tuberculose pouvait-elle aller jusqu'à la guérison complète et à la disparition de la virulence ? Cela reste douteux pour un certain nombre d'animaux qui sont morts sans offrir des lésions macroscopiques évidentes, mais dont les organes n'ont été ni inoculés ni examinés histologiquement. Toutefois dans la première expérience, les organes des 3 derniers survivants de chaque lot ont été inoculés à des cobayes sains. Or ces inoculations ont fourni des résultats négatifs pour 3 cobayes ; deux d'entre eux, appartenant l'un au lot de laboratoire, l'autre au lot de la mer, ne présentaient pas des lésions macroscopiques ; le troisième, appartenant au lot de la campagne, avait dans ses poumons de très petits tubercules grisâtres. Il semble donc que, pour ces 3 animaux au moins, l'infection avait avorté ou avait spontanément guéri.

Il nous a paru intéressant de rechercher quel était, au moment de la mort, le poids moyen des cobayes de chaque lot. Pour la première série, au début de l'expérience, le poids moyen de chaque cobaye était de 554 gr. ; or, au moment de la mort, le poids s'est abaissé à 452 pour le lot du laboratoire, 499 pour celui de la campagne, et 504 pour celui de la mer. Ce sont là, on la voit, des différences peu importantes. Pour la deuxième série, au début de l'expérience, le poids moyen était de 512 gr. Au moment de la mort il était de 516 pour le lot du laboratoire, 511 pour celui de la montagne, et seulement 406 pour celui de la campagne. Mais dans ce dernier lot, l'expérience, restée incomplète, s'est terminée accidentellement beaucoup trop tôt, au bout de 9 mois seulement ; or c'est dans les premières phases que le poids moyen était la plus bas, car dans les 3 premiers mois il n'atteignit que 334 pour le lot de laboratoire, 436 pour celui de la montagne, et 350 pour celui de la campagne.

Bien entendu, la moyenne des poids ne saurait donner l'idée des différences particulières qui pouvaient se rencontre dans chaque lot. Ainsi, certains animaux qui, au terme de l'expérience, n'avaient presque pas de lésions, avaient considérablement engraissé. Dans le lot de la montagne, un cobaye, tué 15½ mois après l'inoculation, pesait jusqu'à 1180 gr. Par contre, un autre, tué en même temps, et dont le poids atteignait le chiffre déjà fort élevé de 950 gr., avait, entre des tubercules pulmonaires, un foie criblé de petits tubercules et une grosse rate pesant 4 gr. 20.

Digitized by Google

Les différences remarquables que présentait l'évolution générale de la tuberculose chez des animaux se retrouvent aussi lorsque l'on entre dans le détail des cas et que l'on considère les lésions des divers organs. Disons tout de suite que l'influence du climat en pareil cas paraît tout à fait nulle et que les différences se trouvent tout aussi accentuées parmi les animaux de chaque lot, soumis aux mêmes conditions d'habitation.

Dans la première série d'expériences, 149 autopsies ont pu être faites ; 74 fois les lésions n'ont pas dépassé la cavité thoracique où, à vrai dire, elles avaient produit des désordres très variables comme forme et comme intensité, certains animaux ne présentant que des tubercules extrêmement petits et discrets, alors même que la mort était tardie, et d'autres ayant, au contraire, des lésions profondes et caséifiées. Chez 75 animaux, les lésions avaient dépassé le thorax et envahi les organs abdominaux, se généralisant au foie, à la rate, qui tantôt étaient fort peu altérés, tantôt littéralement criblés de tubercules.

Dans la deuxième série, 120 autopsies ont été faites. Chez 64 animaux les lésions étaient confinées au thorax, et chez les 56 autres elles étaient plus ou moins généralisées.

Parmi les lésions qui méritent d'être signalées, nous relèverons tout d'abord celles des cavités séreuses. Les pleurésies et les péricardites en forment la majeure partie. Ainsi la pleurésie séro-fibrineuse existait 3 fois et la pleurésie purulente 4 fois ; dans un de ces cas le pus a été examiné bactériologiquement et n'a pas donné des cultures sur les milieux usuels. La péricardite séro-fibrineuse a été notée 6 fois, l'association de pleurésie et de péricardite 3 fois, et dans un de ces cas avec suppuration.

En outre, chez un grand nombre d'animaux les plèvres étaient le siège d'adhérences, notamment du côté de l'inoculation, et le péricarde présentait aussi des adhérences ou des épaississements. Chez 2 animaux, il y avait même une symphyse cardiaque, absolument complète dans un cas. La péritonite a été rencontrée 5 fois, dont 3 avec exsudats purulents.

Plusieurs fois nous avons rencontré des épanchements hémorragiques : hémorragie de péricarde seul chez un animal, de péricarde et de péritoine chez un second, péricardite hémorragique avec épaississement et végétation de la séreuse chez 3 autres. L'un de ces cas était particulièrement remarquable par l'abondance de l'hémorragie : le péricarde distendu avait le volume d'une grosse prune et pesait avec le cœur 25 gr. 50 ; cette lésion avait provoqué de l'arcite et une stase veineuse dans le foie qui offrait l'aspect de foie muscade.

Le péritoine a été, chez 6 animaux, le siège d'une hémorragie. En pareil cas, le point de départ en était la rate, qui apparaissait volumineuse, rouge violacé, friable et déchirée, alors que les autres viscères étaient exsangues : il s'agissait, en somme, d'infarctus spléniques avec ruption et hémorragie. C'est un accident qui est assez particulier à la tuberculose généralisée du cobaye, et qui n'est pas exceptionnel, si nous en jugeons d'après ce que nous avons pu observer dans d'autres séries d'expériences.

La rate présentait d'ailleurs chez les divers animaux des lésions extrêmement variables, comme aspect morphologique. Dans nombre de cas (103) elle

Digitized by Google

ne laissait apercevoir aucune modification, ou bien n'avait subi qu'une augmentation de poids insignifiante, n'excédant pas 1 gramme. Chez 126 animaux, son poids était compris entre 1 et 5 grammes ; chez 27, entre 5 et 10 ; chez 10, entre 10 et 15 ; une fois il atteignit 16 gr. 10, et une autre fois le chiffre énorme de 37 gr. 50, soit plus du dixième du poids de l'animal. Ces hypertrophies considérables étaient dues à des infarctus.

La surface de l'organe était tantôt lisse, tantôt granuleuse ou bosselée, tantôt enfin fissurée par des bandes scléreuses et comme ficelée. Les lésions spécifiques se montraient soit sous la forme de petits tubercules très discrets, soit de nodules plus ou moins volumineux et caséeux, soit de caséification plus ou moins étendue, totale même dans un cas.

Nous n'insisterons pas sur l'état du foie, qui offrait toute la série des altérations qui ont été maintes fois décrites : tubercules à divers états, dégénérescences cellulaires et sclérose.

Mais nous signalerons encore un accident relativement rare, la paraplégie, qui a été rencontrée 5 fois. Elle était due dans un de ces cas à une lésion du rachis qui présentait une déviation angulaire, véritable gibbosité pottique. Mais dans les autres cas, aucune lésion visible ne fut trouvée dans le rachis ni dans la moelle.

Voilà donc toute une série de lésions fort disparates qui se sont développées chez des animaux infectés de même et placés dans les mêmes conditions de milieu et de nourriture. Il est impossible, en présence de ces faits, de ne pas être frappé de l'importance des conditions individuelles, propres à chaque animal, et qui ont déterminé soit une évolution généralisée des processus tuberculeux, soit une prédominance des altérations en certains organes, c'est-à-dire une localisation, soit la forme de la lésion dans chaque organe en particulier. En d'autres termes, le rôle des causes internes dont l'ensemble constitue ce qu'on appelle le *terrain*, apparaît ici d'une façon des plus manifestes.

Si l'on considère que les lésions si variées que nous venons de passer en revue ont pour la plupart leurs homologues en pathologie humaine, on peut comparer les résultats de nos recherches expérimentales avec ce qu'on observe chez l'homme tuberculeux. C'est aussi le terrain qui détermine chez lui la modalité des lésions ; c'est encore le terrain qui régit la marche générale de l'infection. Les chances de contamination tuberculeuse sont, en effet, les mêmes, à peu de chose près, pour un très grand nombre de sujets humains ; la porte d'entrée de l'infection est aussi presque toujours la même. Or, il en est qui résistent ou n'ont qu'une infection minime, qui reste entièrement méconnue et qui avorte, ne se révélant qu'à l'autopsie par la présence de quelques granulations fibreuses. Et le nombre de ces hommes résistantsest relativement considérable, plus grand, certes, que pour nos cobayes. C'est que, d'une part, la réceptivité pour la tuberculose est moindre pour l'homme que pour le cobaye, et d'autre part, que l'inoculation se fait d'ordinaire chez l'homme lentement et par petites doses, et non d'une façon intense et brutale comme dans nos expériences.

Etant donné l'empressement, pour ne pas dire l'enthusiasme universel, en

Digitized by Google

faveur de la création de sanatoria curatifs de la tuberculose, il nous a paru qu'il était nécessaire de fixer avec autant de précision que possible les règles concernant les malades secourus. La méthode expérimentale pouvait seule offrir dans l'espèce toutes les garanties voulues. En procédant sur des centaines d'animaux choisis avec soin elle a fait voir: que l'absence *de tout travail musculaire*, c'est-à-dire *l'immobilisation du sujet*, qu'une *température aussi constante* que *possible* durant les 24 heures et tout le temps du traitement, c'est-à-dire toute l'année, qu'une *alimentation généreuse* sans être excessive, qu'un *air ordinaire non surchargé de microbes*, sans *poussières et tranquille*, constituent les quatre grandes conditions qui donnent une plus grande longévité et des guérisons plus nombreuses.

ON THE COMPARATIVE INFLUENCE OF CLIMATE AND INDIVIDUAL RESISTANCE IN EXPERIMENTAL TUBERCULOSIS.

By Messieurs LANNELONGUE, ACHARD and GALLIARD.

The experiments which we have made in studying the influence of climate in the evolution of tuberculosis inoculated in the pleura of the guinea-pig have taught us that this influence is inconsiderable, and has not the importance which clinical experience has been wont to attach to it.

They have enabled us, in addition, to observe remarkable differences in the progress of the disease and in the nature of the lesions in various animals of the same species, which have been similarly inoculated and submitted to the same conditions of climate and food.

Certain of these animals offered such a remarkable resistance that it would seem that in their cases the infection had not matured, or had been spontaneously cured; some even increased in weight, and the records show that some died quite fat, whilst at the same time exhibiting lesions of generalised tuberculosis.

In nearly half the number of cases the lesions remained localised in the thorax. Affections of the serous membranes, the pleura, pericardium, and peritoneum were extremely frequent, and exhibited the whole series of tuberculous lesions of such membranes met with in man—dry, sero-fibrinous, purulent and hæmorrhagic forms.

A very special feature of tuberculosis in guinea-pigs, of which we have met with six examples, is rupture of the spleen, with profuse hæmorrhage into the peritoneum. The spleen was often affected to such an extent that we have seen it weigh more than one-tenth of the animal.

Most of the lesions met with having their counterpart in human pathology, one can compare these experimental results with what one finds in tuberculous human beings. It is therefore internal causes which together constitute the "soil," far more than external causes and climatic influences, which determine the nature of the lesions and general progress of the disease,

Digitized by Google

The chances of tuberculous infection are almost the same for a large number of human subjects; the onset is also very similar, but the progress of the disease is very different in individuals.

If the number of men who resist it is relatively considerable, and larger than was the case with our guinea-pigs, it is because man is less liable to tuberculosis than the guinea-pig, and he is slowly infected and in small doses and not in wholesale doses, as in our experimental inoculations.

ÜBER DIE PHYSIOLOGISCHEN GRUNDLAGEN DER HOCHGEBIRGSKUREN UND DEREN BEDEUTUNG FÜR DIE TUBERCULOSE.

VON DR. O. AMREIN, *Arosa (Schweiz).*

DIE günstige Einwirkung des Hochgebirges auf den menschlichen Organismus und speziell auf den Verlauf der Lungentuberculose ist eine seit vielen Jahren durchaus anerkannte Tatsache. Wenn ich mir erlaube, hier an dieser Stelle davon zu reden, indem ich eine kurze Zusammenstellung der diesbezüglichen physiologischen Erfahrungen bringe, so ist es namentlich deshalb, weil in der letzten Zeit eine gewisse Strömung sich bemerkbar macht, die—veranlasst durch günstige Resultate in Tiefland-sanatorien—die Tuberculose, wo es ist, selbst inmitten von Städten, heilen will. Es liegt mir ferne, an der grossartigen, volkswirtschaftlichen Bedeutung der Heilstättenbewegung rütteln zu wollen, dass aber die Erfolge vielfach überschätzt worden sind, unterliegt keinem Zweifel. Es äussert sich z. B. *Liebermeister* über diesen Punkt ("Über Lungenschwindsucht und Höhenkurorte," Leipzig, 1898): "Es kommt ja vor, dass ein Kranker auch zu Hause geheilt wird. Es werden auch bei uns in gut geleiteten Anstalten gute Erfolge erzielt. Aber im Hochgebirge werden sie leichter und häufiger erreicht. Wer viele Kranke zu sehen bekommt, die aus den deutschen Heilstätten zurückkehren, der überzeugt sich bald, dass im Hochgebirge die Heilungen, und namentlich die *dauernden* Heilungen, häufiger erreicht werden." *Theodore Williams* hier in London hat in einer Statistik ("The Treatment of Pulmonary Tuberculosis by residence at High Altitudes")* worin er seine Patienten nach gleichen Gesichtspunkten einteilte, constatirt dass die im *Hochgebirge* behandelten vor denen an die Riviera oder auf Seereisen gesandten oder im heimatlichen Klima gebliebenen, den weitaus *Grössten Erfolg* aufzuweisen hatten. (Vergleiche auch: *Egger*, "Über d. Nützen des Hochgebirgsklimas in d. Behandlung d. Lungenschwindsucht.")

Die Hauptcharacteristica des HOCHGEBIRGSKLIMAS, das gerade nach obenangeführter Statistik als das *günstigste* für Lungentuberculose resultirt, lassen Sie mich hier in Kürze schildern. (Vergleiche auch *Römisch*: "Die Wirkung des Hochgebirgsklimas auf den Organismus des Menschen," Essen, 1901.)

---

* *Vide* folgende Seite.

Digitized by Google

Es ist ein Mal die *Luftreinheit*, die wissenschaftlich nachgewiesen ist. Ich führe als bestes Paradigma die häufig citirten Untersuchungen *Miquels* an, nach welchen 10 cm^3 atmosphärische Luft in einer Strasse in *Paris* 55,000 Bacterien, in *Thun* (360 m.) nur 25, und in der Höhe von 2,000 m. gar keine enthalten. Bedingt ist diese Luftreinheit durch die *niedere Lufttemperatur*, von der mit letzterer im Einklang stehenden *Schneedecke* (Mitte November bis Mitte April), durch den Licht- und Sonnenreichtum. Ferner spielen dabei eine Rolle: eine spärliche Bevölkerung, das Fehlen von Fabriken, Eisenbahnen. Auch halte ich für wichtig, dass die Wohnstätten von Menschen und Tieren nicht dicht aneinander gebaut sind, sondern zerstreut und einzeln stehen, wie es z. B. in Arosa durch die Terrainverhältnisse nicht anders möglich ist. Was nun die *niedere Lufttemperatur* anbetrifft (die, wie ich eben sagte, viel zur Reinheit der Luft beiträgt), so ist für das Hochgebirge characterisch die *gleichmässige Kälte im Winter und die geringe Sommerwärme*.

RESULTS OF DIFFERENT CLIMATES COMPARED (nach Theodore Williams).

	No. of Patients.	Average length of Residence.	First Stage.	Second and Third Stages.	Bilateral affection.	Results.						
						General.			Local.			
						Improved.	Stationary.	Worse (incl. deaths).	Arrest.	Improved (incl. Arrest).	Stationary.	Worse.
		months	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
High altitudes .	247	12·2	65	35	37	83·4	2·02	14·57	42·5	75·5	5·3	19·1
Sea voyages .	65	1·6 Average of voyages.	63	37	37	77	...	21·56	7·7	53·3	10·7	33·8
Riviera . . .	210	9	59	41	36	65	10	24·8	5·9	36·6	17·8	45·6
Home climates .	292	9·7	58	42	42	63·7	8·21	28	2	38·9	20	41·1

Im Winter bewegt sich die Temperatur um wenige Grade um den Gefrierpunkt herum; in den Nächten sinkt sie häufig sehr tief. Ich verweise diesbezüglich wie für die folgenden Ausführungen auf diese Tabellen, welche ich nach den mir von der meteorolog. Station in Arosa (Dr. Janssen) gütigst überlassenen Aufzeichnungen zusammengestellt habe. Dass es im Sommer im Hochgebirge sehr heiss sei, ist eine noch viel verbreitete irrige Aussicht. Aus den angeführten Tabellen ersehen sie die Unrichtigkeit derselben. Auch erwähne ich noch, dass unsere Hochgebirgskurorte alle zuerst als *Sommerfrischen* bekannt wurden. Dass auch starke Kältegrade im Hochgebirge nicht empfunden werden wie im Tiefland (unsere Kranken liegen bis $-20°$ und mehr bei angenehmstem Befinden bis in die Nacht auf den Balkons) rührt von der *ausserordentlichen Trockenheit der Luft her*. Sie finden unter den meteorol.

Digitized by Google

TAB. I.—Meteorologische Beobachtungen in Arosa.

1900. 1901.

	Luftdruck bei 0° C. (Air pressure 32° F).		*Lufttemperatur* (Temperature).		*Relative Feuchtigkeit* (Relative humidity).	*Bewölkung* (Clouds).	*Niederschlagsmenge* (Rainfall).		*Zahl der Tage mit* (Number of the days with)		*Zahl der Tage mit* (Number of the days with)			*Sonnenscheindauer in Stunden.*	Sonnenscheindauer in Stunden.
	Mittel (mean).		Mittel (mean).		Mittel (mean).	Mittel (mean).	Summa.		*Regen & Schnee* bis 0·3 mm. (under 0·012 inch).	*Regen & Schnee* bis 1 mm. (under 0·04 inch).	*Schnee* (Snow).	*Hagel* (Hail).	*Gewitter* (Thunderstorm).	(Sunshine) (hours).	(Sunshine) (hours).
	mm.	inches.	Celsius.	Fahr.			mm.	inches.							
January .	606·6	23·88	−4·3	24·3	67	70	136	5·44	21	21	21	...	...	42·0	119·2
February .	603·0	23·74	−2·5	27·9	62	62	110	4·40	16	14	16	...	...	81·1	108·3
March . .	604·5	23·79	−5·4	22·3	62	60	93	3·72	16	15	16	...	...	123·2	98·3
April . .	608·8	23·96	+0·3	32·5	66	66	92	3·68	12	11	12	2	...	121·9	149·6
May .	609·1	23·98	4·6	40·3	73	75	130	5·2	16	15	12	1	...	98·1	
June . .	612·7	24·01	9·8	49·6	66	63	80	3·2	10	10	1	1	2	121·4	
July . .	615·1	24·21	12·2	54·0	71	54	170	6·8	16	15	2	...	6	174·0	
August .	613·4	24·14	9·8	49·6	76	59	233	9·3	17	15	0	...	3	167·4	
September	616·4	24·26	10·7	51·3	64	46	35	1·4	10	8	0	...	...	176·4	
October .	616·4	24·26	4·8	40·6	57	39	37	1·5	8	8	3	1	...	156·5	
November.	609·0	23·97	+0·3	32·5	64	52	64	2·6	11	9	10	...	...	71·2	
December .	614·7	24·19	−0·9	30·4	51	42	69	2·8	9	7	9	...	...	88·3	

(Nach den Beobachtungen der Meteorol. Station in Arosa; Dr. Janssen.)

Digitized by Google

TAB. II.—Vergleichung zwischen Arosa und Zürich.

Windverteilung im Jahre 1898.																		Sonnenscheindauer, 1898 (Sunshine).		
	Zürich.									Arosa.									Zürich.	Arosa.
Month.	N.	N.E.	E.	S.E.	S.	S.W.	W.	N.W.	Calmen.	N.	N.E.	E.	S.E.	S.	S.W.	W.	N.W.	Calmen.	Month.	Stunden (hours).	Stunden (hours).
January . . .	7	7	1	3	1	3	1	4	66	...	1	2	1	2	2	33	5	47	January . . .	39·0	146·5
February . . .	3	2	5	1	6	15	21	5	26	...	1	11	4	2	...	9	1	56	February . . .	80·3	76·5
March	4	9	9	1	2	12	9	7	40	...	...	4	11	...	4	16	1	57	March	93·7	132·7
April	7	13	6	1	3	5	13	8	34	...	...	10	3	1	9	12	...	55	April	142·7	140·2
May	4	9	2	2	6	14	15	17	34	...	...	10	11	4	13	3	...	52	May	36·1	150·9
June	2	6	7	5	11	7	13	3	36	...	1	13	5	6	1	2	...	62	June	169·8	130·4
July	5	10	3	1	6	12	9	11	36	...	2	25	5	3	...	1	...	57	July	238·0	199·6
August . . .	2	5	5	3	2	5	10	6	55	...	0	19	10	2	2	...	...	60	August . . .	304·6	275·1
September . .	4	17	5	5	2	3	8	8	38	1	1	15	13	2	2	2	...	54	September . .	242·6	240·7
October . . .	2	9	5	4	6	6	11	9	41	1	1	22	5	3	5	1	4	51	October . . .	70·3	138·3
November. . .	8	11	9	7	2	3	7	4	39	1	1	9	7	1	12	10	2	47	November. . .	87·8	97·0
December. . .	3	3	6	2	9	8	12	4	46	...	1	4	...	...	5	20	2	61	December. . .	50·6	119·3
The whole year .	51	101	63	35	56	93	129 (Localwinde.)	76	491	3	9	144	75	26	55	109	15	659	The whole year .	1595·5	1847·2

(Nach den Annalen der Schweizer. Meteorolog. Centralanstalt, 1898.)

Digitized by Google

Aufzeichnungen auch die entsprechenden Werte (*relative Feuchtigkeit*). Dafür spricht auch die Art unserer Einheimischen, ihr Fleisch statt im Rauchfang an der blossen Luft zu trocknen, und ferner (in unangenehmer Weise) das Springen des Holzes, das Austrocknen der Cigarren und das Rissig-werden der Wände, etc.

Neben der Luftreinheit ist für das Hochgebirge characteristisch der *Sonnenreichtum*. Die Einwirkung der Sonnenstrahlen ist bei der reinen und trocknen Luft ein ganz enormer. Der geringe Gehalt der Atmosphäre an Wasserdampf bringt es mit sich, dass eine viel geringere Absorption der Sonnenstrahlen stattfindet als im Tal. Weil Neben- und Wölkenbildung seltener sind (vide Tabelle I und II) so ist diese *Insolation* eine andauerndere und ergiebigere.

Fernerhin ist ein Hauptfactor unseres Klimas der *niedere Luftdruck* und die damit verbundene *Verminderung des Sauerstoffgehaltes der Luft*. (Barometerstand in Arosa im Mittel 610 mm.; vergl. Tab. I d. meteorol. Beobachtungen.) Weiter ist hervorzuheben die *Windstille*. In dieser Beziehung sind bis jetzt einzig *Davos* und *Arosa* (von den Graubündnerbergen) als besonders begünstigt bekannt; speziell *Arosa* ist durch Bergvorsprünge vor lokalen und durch die nahen Bergketten vor obern Winden sehr geschützt. Ich verweise diesbezügl. auf Tab. II, wo ein Mal (im Vergleich mit Zürich, z. B.) die hohe Zahl der *Calmen* auffällt und speziell der grosse *Schutz* vor den *scharfen Nord- und Nordostwinden*.* Einzig der *Föhn*, der spezifische, warme Wind der Nordalpentäler spielt ab und zu eine Rolle, der Wind, der durch das schnelle Herabstürzen von den kalten Bergkämmen unter erhöhten Drucke sich erwärmt und lau und schwer in die Täler fliesst.

Was für einen Einfluss diese in obigem kurz zusammengefassten Hauptfactoren des Hochgebirgsklimas auf den Organismus und die Körperfunctionen ausüben, lassen Sie mich dartun, indem ich mich auf die entsprechenden

Physiologischen Untersuchungen

beziehe. Es sind das in erster Linie ein Mal die *Blutuntersuchungen* (ich beschränke mich in der Hauptsache auf die Arbeiten von Schweizerforschern). Nachdem zuerst *Viault*† (1890) in Marococha (Peru) in einer Höhe von 4,392 m. über Meer entdeckt hatte, dass beim Menschen nach dem Gelangen an einen Ort von erheblicher Höhe sich die *Zahl der roten Blutkörperchen vermehre*, und um so mehr, je grösser die Höhendifferenz des frühern und des gegenwärtigen Aufenthaltes sei, folgte eine Reihe wertvoller Arbeiten über diese neue Entdeckung, zunächst von *Basel* aus (*Mieschersche Schule*).‡ Es war in *Arosa*, wo *Egger* (in einer Höhe von 1,800 m.) bei 27 Personen

---

* Die angegebenen Winde sind *Localwinde*.

† Auf Beobachtungen von *P. Bert* hin, der zu Blutuntersuchungen aufforderte, um die Anpassungsfähigkeit des Menschen an die Hochgebirgs-einwirkung zu erklären.

‡ "Die histochemischen und physiolog. Arbeiten von *Miescher*," Bd. ii, Leipzig, 1897.

Digitized by Google

(Männern und Frauen) in durchschnittlich 14·5 Tagen eine Vermehrung der roten Blutkörperchen von 5·4 auf 6·3 Millionen (d. h. um 16 %) fand.* Bei der Rückkehr in die Ebene constatirte Egger, wie auch schon Viault, eine Verminderung der Zahl der roten Blutkörperchen, und zu gleichen Resultaten gelangten später *Mercier* in Arosa, *Kündig* in Davos und *Römisch* in Arosa. Letzterer† fand "eine Zunahme der *Zahl der roten Blutkörper*, die bei 21 acclimatisirten Männern im Durchschnitt 6·497000, bei 9 acclimatisirten Frauen im Durchschnitt 5·459000 betrug. Diese Zunahme liess sich bei 9 Personen (6 Männern und 3 Frauen) zwischen der ersten und zweiten Untersuchung in Arosa nach durchschnittlich 17 Tagen Aufenthalt daselbst als durchschnittlich 11 % berechnen." Bei 5 Personen (3 Männern und 2 Frauen) die er zuerst im Tiefland und darauf in Arosa untersuchen konnte, betrug die Vermehrung durchschnittlich 26 % nach eingetretener Acclimatisation in Arosa. Bei dem Verlassen von Arosa trat regelmässig eine Abnahme der Zahl der roten Blutkörper ein. Bei Tuberculösen war die Zunahme der Zahl der roten Blutkörper in Arosa im Durchschnitt noch etwas bedeutender als bei Gesunden. Der gleiche Autor macht zudem aufmerksam auf die "grossen individuellen Verschiedenheiten"; die Vermehrung ist nach ihm, z. B., eine erheblich geringere bei chlorotischen und anämischen Personen.

Von den Einwendungen, die gegen die citirten Untersuchungen gemacht wurden, war ein Mal derjenige, dass die Vermehrung der roten Blutkörper nur eine *scheinbare* sei. Es sollte sich um "einen vorübergehenden Reizzustand des Gefäss-systems" handeln; *Egger* hatte schon diesen Einwand widerlegt, indem er zeigte, dass es eine *dauernde Vermehrung* sei, indem er sie bei 10 Gesunden Eingebornen in Arosa constatirte; das gleiche fand *Römisch* bei 5 seit Jahren in Arosa Niedergelassenen. Ebenfalls *Egger* sah die Vermehrung sowohl im Blute aus den Arterien als aus den Hautcapillaren (an Kaninchen). demzufolge es sich nicht um eine "*abnorme Verteilung des Blutes*" handeln kann. Dass auch nicht eine "*Eindickung des Blutes durch gesteigerte Wasserabgabe des Körpers* in Frage kommt, zeigte Egger an 2 Kaninchen, denen er in Basel Blut entzog und dessen fixe Bestandteile bestimmte, und die er nachher in Arosa 3 Wochen lang genau wie in Basel fütterte, dann die gleiche Quantität Blut entnahm, aus welchem *Miescher* in Basel ebenfalls die fixen Bestandteile ermittelte und dabei auf keinen erheblichen Wasserverlust stiess. Dagegen spricht auch unsere Erfahrung, dass bei den ins Hochgebirge kommenden in der Acclimatisationsperiode in der Regel eine *Gewichtszunahme* eintritt, und nicht eine Gewichtsabnahme, wie sie nach *Czerny* bei einer solchen Polycythaemie in Folge Bluteindickung statthaben sollte ('Arch. f. Exp. Pathologie und Pharmacol.,' Bd. xxiv). Gegen die Annahme eines *dauernden Contractionszustandes*" der Blutgefässe spricht das Verhalten des *Haemoglobins*, dessen Gehalt im Blut (nach *Viault*, *Egger*, *Wollf* und *Kappe*, *Römisch*, *Suter* und

---

* "Beobachtungen an Menschen und Kaninchen über d. Einfluss des Klimas von *Arosa* auf das Blut," 1896. 'Archiv. f. Experiment. Path. und Pharmacol.,' Bd. xxxix.

† "Beiträge zur Frage der Einwirkung des Höhenklimas auf d. Zusammensetzg. d. Blutes." "Festschrift d. Dresdner Stadtkrankenhauses."

Digitized by Google

Jaquet) im Gebirge steigt, allerdings erst später als die Vermehrung der rot-Blutkörper (davon er abhängig ist).

Ein letzter Einwand war der, dass das "unrichtige Resultat" der Blutkörper-Vermehrung einer mangelhaften Zählkammer zuzuschreiben sei (*Gottstein*, 'Berl. kl. Woch. Schrift,' 1898, 21/22). *Meissen* hat den gerügten Übelstand, dass in die Thoma'sche Zählkammer die Luft keinen Eintritt habe, durch einen radiären Einschnitt am Rande derselben (wo das Deckglas aufliegt) ausgeschaltet. Es ergaben aber die Versuche mit dieser *Meissenschen Schlitzkammer* (*Turban, Soholowsky, Kündig und Karcher*) dass durch sie die Blutkörperchenzahl nicht beeinflusst wird. Worauf die Vermehrung der roten Blutkörper beruht, ist trotz verschiedener Hypothesen nicht sicher bekannt. *Miescher* wollte in dem O.-Mangel den spezifischen Reiz zur Neubildung sehen, *Ehrlich* und *Lazarus* erklären nach den Befunden *Köppes* "die anfängliche Vermehrung durch Abschnürung und Teilung," und das Steigen des Haemoglobingehaltes durch den erst nachher eintretenden Process die Neubildung roter Blutscheiben.

Ausser der Vermehrung der Zahl der roten Blutkörperchen und des Haemoglobingehaltes durch die Einwirkung des Hochgebirges sind noch constatirt worden: Eine *Erhöhung des spezifischen Gewichtes* des Blutes (Müntz*), das Auftreten von *Microcyten* in der Acclimatisationsperiode (*Viault* und *Mercier*), und das Vorkommen von *Kernhaltigen roten Blutkörperchen* im Menschenblut unter der Einwirkung des Höhenclimas (*Schauman* und *Rosenquist*, 'Arch. f. d. ges. Physiologie,' Bd. 68).

Eine *Abnahme der Zahl der weissen Blutkörper* nach erfolgter Acclimatisation im Hochgebirge haben *Schröder* und *Meissen* ('Münch. Med. Woch.-Schrift,' 1897, No. 23) betont, während die Untersuchungen von Römisch (loc. cit.) keine wesentliche Änderung in dem Verhältnis von roten zu weissen Blutkörpern ergaben.†

Von welchen obengeschilderten *Klimatologischen Factoren* diese Veränderungen des Blutes im Hochgebirge abhängt, darüber hat *Jaquet* in Basel Untersuchungen angestellt ("Recherches sur l'action physiologique du Climat d'Altitude," 'Semaine médicale,' 26 sept. 1900). Er kam dabei zu folgenden Resultaten: *Die Temperatur* hat *keinen* Einfluss auf die Zusammensetzung des Blutes. Je 6 Kaninchen wurden je bis 13–16° C. und 2–5° C. unter gleichen Bedingungen gehalten und nach 6 Wochen auf ihr Blut untersucht. Der Hauptfactor nach ihm ist der *niedrige Luftdruck* (experimentell an Apparaten und durch Vergleichungen zwischen Basel und Davos ermittelt), und er sagt:—"Eine Verminderung des Druckes um 100 mm. Quecksilber genügt, um ohne Miteinwirkung eines andern Factors des Höhenclimas die Veränderungen in der Blutzusammensetzung zu erklären."

---

* 'Compt. rend. de l'Acad. d. Scien.' 1891.

† Vergleiche noch: "Untersuchungen über d. Einfluss des Höhenclimas auf die Beschaffenheit des Blutes," Arbeiten von *Veillon, Egger, Karcher, Suter* und *Jaquet*, in *Mieschers* "Histochem. und physiol. Arbeiten," Bd. ii.

Digitized by Google

Der *Trockenheit der Luft* schreibt er keinen in Betracht kommenden Einfluss in dieser Beziehung zu.

Nachdem *Schumburg* und *Zuntz* die Erklärung der Blutveränderungen in der *intensiven Sonnenbestrahlung* erblickten ("Zur Kenntnis der Einwirkung des Hochgebirges auf den menschl. Organismus": 'Arch. f. d. ges. Physiol.,' 1896, Bd. lxiii.) und *Marti* ("Wie wirken chem. Reize und Belichtung auf die Bildung der roten Blutkörperchen?" "Verhandlgn. d. Congresses f. innere Medizin," 1897), bei Ratten in der Dunkelheit eine Abnahme, im Lichte eine Vermehrung der rot-Blutkörper gefunden hatte (ein Befund dem *Schönenberger* widersprach ["Einfl. d. Lichtes auf. d. hum. Organismus," Berlin, 'Diss.,' 1898]), brachte *C. Meyer* (Basel) die einwandsfreien Ergebnisse seiner vergleichenden Untersuche über diesen Punkt in Basel und Davos und constatirte ("Einfluss d. Lichtes im Höhenklima," 1900) dass die Abwesenheit des Lichtes keinen Einfluss auf die Wirkung des Höhenclimas ausübe.

Nach diesen Blutuntersuchungs-Arbeiten beschäftigte sich denn *Jaquet* in Verbindung mit *Stächelin*, mit

Stoffwechsel-untersuchungen.

Die bis jetzt gefundenen Resultate ("Vergleichungen zwischen Basel und dem Chasseral," 1900; 'Semaine médicale,' 26 sept. 1900) sind diese: dass die Vermehrung der roten Blutkörper und das Haemoglobingehaltes des Blutes begleitet sind von einer deutlichen *Verminderung der Stickstoffausscheidung* des Körpers, und es ist nach den Arbeiten der genannten Autoren anzunehmen, dass "die *Wirkung des Höhenclimas sich nicht auf die haematopoetischen Organe erstrecke sondern dass vielmehr sie sich auch über andere Elemente ausdehne* bei denen sie durch teilweise *protoplasmatische Regeneration eine neue Lebenskraft und Widerstandsfähigkeit* hervorbringe." Nach inzwischen erschienenen Arbeiten derselben Autoren ('Semaine médicale,' 1901, No. 28) ergaben sich unter dem Einfluss des Hochgebirgsklimas: Eine Retention von Stickstoff, ein vermehrter Verbrennungsprocess im Körper und eine Zunahme des Respiratorischen Quotienten (ein grösserer Verbrauch von Sauerstoff und grössere Ausgabe von Kohlensäure), ein Befund, der überraschend ähnlich demjenigen in der Reconvalescenz nach schweren, fieberhaften Krankheiten ist. Was nun die

Klinisch-practischen Beobachtungen

bei d. Hochgebirgskuren anbetrifft (die früher bekannt geworden sind als die oben erwähnten, die erklärenden physiologischen Überlegungen und Untersuchungen) so lassen sie mich zunächst kurz auf diejenigen bei Lungentuberculose eintreten.

Sehr bald, nachdem unsere Lungenpatienten in unsere Bergwelt gekommen sind, ist meist eine ganz bedeutende *Besserung im Allgemeinbefinden* zu constatiren (abgesehen von sehr anaemischen und nervösen Personen, bei denen sich während der Acclimatisationszeit hin und wieder Beklemmungen, Berg-

Digitized by Google

klopfen u. s. w. einstellen). Die *dünne reine Luft* und der stetige Aufenthalt in derselben (Freiluftkuren nach Brehmer-Dettweiler) regen den *Appetit* an, und dieser—welcher oft so lange darnieder lag, steigert sich, oft zu imponirender Stärke, auch bei Bettlägerigen oder sich sonst ruhig Verhaltenden. Dadurch ergibt sich eine baldige *Gewichtszunahme*, was beides auf die Kranken und ihre Psyche sehr wohltätig einwirkt. In der trocknen Luft tritt überraschend schnell das *Aufhören der lästigen und schwächenden Nachtschweisse* ein und namentlich (oft nach lange andauerndem Fieber zu Hause) erfolgt eine fortschreitende *Entfieberung*. Die zu Bette liegenden Fieberkranken sind auch dort in fortwährendem Contact mit der reinen, guten Luft, die durch die offenen Fenster und Balkontüren zu ihnen eintritt.

In der ersten Zeit können sich *Husten und Auswurf etwas vermehren*: meistens tritt aber schon sehr bald, ohne Medicamente, ein *Nachlassen des Hustenreizes* und dadurch ein *guter, ungestörter Schlaf* ein.

Am *Lungenbefunde* ist allmälig ein *Abnehmen* und *Verschwinden* der *Rasselgeräusche* zu constatiren, ein *Austrocknen* der erkrankten Partien, unter Verschwinden des Sputums, das Auftreten von reinem *Narbenahnen*, unter *Schrumpfungvorgängen*, also ein stetig *vorwärtsschreitender Heilungsprocess*.

Nicht zu übersehen ist die *psychische Beeinflussung* der Kranken durch die helle *Sonnenpracht*, wie sie eben das Tal nicht kennt. Dieser lachende Sonnenschein, im Verein mit der Erhabenheit des Bergwelt, gibt dem nach Gesundheit Ringenden frische Hoffnungsfreudigkeit und Lebensmut. So erinnere ich mich selbst als einer der schönsten Moments, an den neuen kräftigen Pulsschlag des Lebens, als ich als Patient nach Arosa kam und dort Erholung von Siechtum und Heilung suchte und fand.

Neben diesen günstigen Resultaten gibt es natürlich auch Fälle, wo eine Besserung leider nicht mehr eintritt; es sind das ein Mal die zu weit *ausgedehnten Lungenerkrankungen*, wo ungeachtet aller Therapie und jeden

Von 120 im Hochgebirge Behandelten hatten daselbst Lungenblutungen:
Total . . 12 = 10 %.

	1 Stadium.	2 Stad.	3 Stad.	Total.
Von diesen 120 hatten zuvor im Tiefland Lung. Blutungen . .	22 (=18·3 %)	20 (=16·6 %)	8 (=6·6 %)	50 (=41·6 %)
Von den 50 Fällen mit Haemoptoe im Tiefland wiederholten sich Lungenblutungen im Hochgebirge bei . .	4 (=18·2 %)	4 (=20·2 %)	2 (=25·7 %)	10 (=20 %)
Erst im Hochgebirge hatten Lungenblutungen	2 (=1·7 %)	...	...	2 (=1·7 %)

Digitized by Google

Climas das Werk der Zerstörung seinen Gang schreitet; es sind oft auch *individuelle* Factoren, die der Arzt zu beachten hat, und damit komme ich zu den *Contraindicationen* für Kuren im Hochgebirge. Diesbezüglich sind anzuerkennen (nach *Egger*: "Üb. d. Nutzen d. Hochgebirgsklimas in d. Behandlg. d. Lungenschwindsucht," 'Zeitchrift f. diätet. und physical. Therapie,' 1899, Bd. iii, 2):

Ausgesprochene Tendenz zu unaufhaltsamem Fortschreiten d. Lungenerkrankung, ausgebreitete Fälle mit Zerfall, hectischem Fieber und Marasmus; zu grosse Beschränkg. d. Atmungsoberfläche infolge ausg. Erkrankg. Ausgedehnte Larynxerkrankungen, Complicationen d. Lungentuberculose mit hochgr. Emphysem, Nierenerkrankungen, organ. *Herzleiden, Intestinaltuberculose, psychische Abnormitäten.*

Fieber allein darf nicht als Contraindication gelten; ich habe oben darauf hingewiesen, dass eine Entfieberung im Gebirge eher eintritt als im Tieflande. Unbegründet ist auch die Annahme, dass die *Neigung zu Blutungen* einen Aufenthalt im Höhenclima ausschliessen soll.

Ich erlaube mir diesbezüglich in folgender Tabelle eine Zusammenstellung der *Blutungen* aus meiner Praxis vor und während des Aufenthaltes im Hochgebirge zu geben.

Es ergibt sich daraus (wie es auch schon früher von *Spengler, Peters, Egger*, constatirt wurde) dass Lungenblutungen im Hochgebirge nicht nur nicht häufiger, sondern *seltener* sind als im Tieflande.

Nach diesen Ausführungen ist meine Ansicht diejenige, dass die Hochgebirgskuren (unter strenger ärztlicher Controlle) das Beste sind, was es bis jetzt zur Heilung der *Lungentuberculose* gibt, und es sollte nach meiner Überzeugung jeder Lungenkranke, wenn er transportfähig ist und keine der genannten Contraindicationen aufweist, *sofort* in das Gebirge gesandt werden, selbst fiebernd, ohne lange Zeit im Tal nutzlos und schadenbringend zu verlieren, und auch zu *jeder Jahreszeit.*

Gewiss ist es der *Winter* vor Allem, der günstig einwirkt, schon ein Mal wegen der Umgehung dieser meist ungesunden Jahreszeit im Tieflande. Aber auch der *Sommer* übt die gleichen Wirkungen aus, bleiben ja die Hauptfactoren stets dieselben: niedrige Temperatur, niedriger Luftdruck, Reinheit und Trockenheit der Luft und Sonnenreichtum (vide die meteorol. Tabellen). Zudem spenden dann die ozonreichen Fichtenwälder (wie wir sie z. B. in Arosa nicht genug bewundern können) würzige Luft und Kühlung.

Die *Furcht vor der Schneeschmelze* ist ferner eine unbegründete. Es ist nach *Turbans* Statistik ("Beiträge zur Kenntniss der Lungentuberculose," Wiesbaden, 1899) ein schädlicher Einfluss jedenfalls ausgeschlossen ("in den Jahren 1889–1896 waren im ganzen Jahr durchschnittlich 13·5% aller Kranken bettlägerig, im März und April nur 11·7 ‰, obwohl gerade in diesen Monaten in allen Klimaten Mitteleuropas leicht Rückfälle von Tuberculose und Katarrhen vorkommen"). Zudem vollzieht sich (gerade z. B. in Arosa) der Vorgang der Schneeschmelze ganz allmälig, so dass immer gangbare Wege vorhanden sind. Die Patienten werden in ihrer Liegekur und in ihrem Spazierengehen gar nicht, oder nicht stark, beeinträchtigt.

Digitized by Google

Gerade das ist ein ungeheurer Vorzug vor den südlichen Kurorten, dass im Hochgebirge unabhängig von Wetter und Jahreszeit mit gleichen Erfolgen Kur gemacht werden kann, ganz abgesehen davon, dass die aus dem Hochgebirge zurück-kehrenden Geheilten oder Gebesserten den Winter im Tiefland und Norden ertragen können und nicht denselben immer wieder, wie die an den Süden gewohnten umgehen müssen.

Zum Schlusse möchte ich nur noch flüchtig den *Einfluss des Hochgebirges auf die* CHIRURGISCHE TUBERCULOSE erwähnen; ich beziehe mich dabei namentlich auf die treffliche Arbeit *Wölfflins* ('Dissert.,' Basel, 1899), welche uns die interessanten Schlussfolgenungen mitteilt (im Verein mit Bernhard nach Beobachtungen im Engadin gewonnen) dass die chirurgische Tuberculose im Hochgebirge einen reinern und bessern Wundheilungsverlauf und eine ungestörtere Reconvalescenz *post operationem* nehme; dass die operativen Eingriffe zu gunsten der *conservativen* Methode mehr eingeschränkt werden können als im Tiefland. Namentlich würden chronische Lymphome, tuberculöse Knochen- und Gelenkerkrankungen, sowie die Urogenitaltuberculose, wahrscheinlich auch der Lupus, in günstigster Weise vom Höhenclima beeinflusst. In meiner eigenen Praxis sah ich eine überaus schnelle und definitive Heilung nach Auskratzung einer Caries der IX. Rippe mit Abscedirung, und in sieben Fällen ein Zurückgehen und Verschwinden multipler Lymphome ohne Operation. Wölfflin empfiehlt (worauf ich auch besonders aufmerksam mache) das Hochgebirge für *scrophulöse und belastete Kinder* zur *Prophylaxe.* Dieser Punkt fängt übrigens an, mehr gewürdigt zu werden, nachdem ein Mal die Furcht vor Infection durch das roborirenden Klima selbst und die an unsern Kurorten herrschenden Disinfectionsvorschriften überwunden wurde.

THE PHYSIOLOGICAL PRINCIPLES OF THE HIGH-ALTITUDE TREATMENT AND THEIR IMPORTANCE IN TUBERCULOSIS.

BY O. AMREIN, M.D., *Arosa, Switzerland.*

MR. PRESIDENT, LADIES AND GENTLEMEN,—My reason for speaking before this meeting about the influence of a high-mountain climate on the human organism generally, and on tuberculosis in particular, recognised as very important for many years, is that there has lately been an agitation, based on good results obtained in lowland sanatoria, to cure tuberculosis wherever it exists, even in the midst of crowded towns. I do not wish for one moment to cast a doubt on the usefulness of these sanatoria in the lowlands, but it appears to me that too much has been made of the results obtained in them. This is the opinion, too, of *Liebermeister*, *Erb*, and especially of *Dr. Theodore Williams*, who has shown that those of his patients who were sent to the high mountains returned home showing better results than those who were sent to the Riviera or ordered sea voyages of considerable extent.

Digitized by Google

I some years ago assisted as doctor in a Swiss lowland sanatorium, and, notwithstanding this establishment is an extremely good one, I see much better results now in the high mountains.

I can only mention quite shortly the characteristics of a high-mountain climate; you will find all details in the Transactions. A reference to the tables which I have constructed from meteorological observations made at Arosa will show you the results of these observations of each month in 1900.

I mention now only the *purity of the air* and its freedom from bacteria and other germs. The *low temperature:* On this point I must remark, it is an error to suppose, as some do, that the summer-time is a hot season in the mountains. The tables will show that the highest average temperature in July is only 54° F., and the places in the high mountains which are now famous as health resorts were first known, on account of their moderate summer temperature, as pleasant summer residence places. Then I mention the extreme *dryness of the air*, the *richness or great actinic power of the sunshine* and therefore an *intense insolation*, the *low barometric pressure* and therefore a smaller quantity of oxygen, and the *quietness of the air*, or freedom from winds. In this respect Arosa and Davos are especially sheltered from winds, and pre-eminent among the health resorts of the Canton des Grisons.

I must say that I think it of great importance that a locality selected as a cure place (besides these climatological characteristics) should not have a large population, and should be free from railways and such manufactories as might pollute the air with noxious odours and fumes, and that the buildings should be so far apart as to permit a free circulation of the air between them. At Arosa, for example, the hilly and uneven nature of the land on which the village-town is built renders overcrowding of any kind impossible.

What influence the above-mentioned attributes of a high climate have on the organism and functions of the human body may be learned from the scientific and physiological observations and experiments to which I will now shortly refer.

First as to *Examinations of the Blood* (I give here only a short extract):

After *Viault's* observations—who found in 1880, in Peru, 14,000 feet above sea-level, an *increase in the number of the red corpuscles* in the blood of those persons who came from a lower altitude to a high one—many important papers on the same subject appeared—by *Egger* at Arosa, *Kündig* at Davos, and by *Mercier* and *Römisch* at Arosa—showing similar results (an increase of 16·0 to 20 per cent. in the mean) after about a fortnight of staying at the higher elevations, the increase being *greater* in the case of *tuberculous* subjects. All objections have been met, such as that the increase might be due only to a temporary contraction of the vessels, or to a thickening of the blood, or to a permanent contraction of the vessels. In addition to this increase in the number of the *red corpuscles*, there is found an increase in the amount of hæmoglobin present and a greater specific gravity. The appearance of microcytes, of red corpuscles with nuclei, and a diminution of the white corpuscles, is not yet satisfactorily established.

He also met the objection that the increase might be due to a thickening

Digitized by Google

of the blood by loss of water by experiments on rabbits, made first at Bâle, and then after keeping them three weeks at Arosa he found that no difference in the fixed constituents of the blood could be shown. The increase of the hæmoglobin shows also that the increase of red corpuscles is not due to a *permanent contraction* of the vessels. The objection that the most used Thoma-Zeiss apparatus gave results which were erroneous, not being independent of the pressure of the air, is groundless; it is shown by the fact that similar results are obtained with the new apparatus of Meissen, into which the air can enter.

In addition to this increase in the number of red corpuscles and in the amount of hæmoglobin present, it is found that the blood at high-mountain altitudes has a greater specific gravity than it has in the lowlands. The appearance of microcytes and red corpuscles with nuclei and a diminution of the white corpuscles is not yet satisfactorily established.

Jaquet and *Suter*, of Bâle, have made experiments with a view of finding out by what climatological elements this change in the composition of the blood is caused. They find that neither the low temperature nor the extreme dryness of the air has any influence, but that the change is due to the *low pressure of the air;* and *C. Meyer*, of Bâle, proved, in opposition to the views of Schumburg and Züntz, that the changes in the composition of the blood are quite independent of the absence or presence of light.

Consequent on these observations on the blood, *Jaquet*, in collaboration with *Stächelin*, further found in high altitudes a diminution of the elimination of *nitrogen*, and the same author says that the changes in the organism produced by the influence of high altitudes extend beyond the hæmatopoietic system, and by a partial protoplasmic regeneration confer on the other elements of the organism a new vitality and power of resistance to disease.

According to some very recent and just published experiments, the same authors have found an increase of the process of combustion in the body and an increase of the respiratory *quotient*—that is to say, an increased consumption of oxygen and a greater expiration of carbonic acid.

These facts are astonishingly like those which are observed during convalescence after grave febrile diseases, and it is to be hoped that this interesting point may receive further attention.

Now, as to the *clinical observations*, I come in the first place to—

The Tuberculosis of the Lungs.

Very soon after the arrival of our patients in the high-mountain health-resorts a great improvement in their subjective state may generally be observed; anæmic and nervous people, however, suffer from palpitations and sleeplessness, etc., during the time of their acclimatisation. The continual living out in the open and thin air increases the appetite often to an astonishing extent, and in consequence there results an increase in the bodily weight which has a very good psychical influence on the patient.

The extreme dryness of the air puts a stop in a very short time to the

Digitized by Google

I.—Meteorological Table from Arosa.

	1900.														1901.
	Barometric pressure.		Temperature of the air.		Relative humidity.	Cloudiness.	Rainfall (Amount).		Number of the days with		Number of the days with			Average Sunshine	Average Sunshine
	mean		mean												
	at 0° C mm.	32 F (inches).	C°.	F°.	mean	mean	à mm.	(inches).	Rain and snow up to 0·3 mm. (0·01 inch).	Rain and snow up to 1 mm. (0·04 inch).	Snow.	Hail.	Thunderstorms.	in hours.	in hours.
January . . .	606·6	23·88	− 4·3	24·3	67	70	136	5·4	21	21	21	..	..	42·0	119·2
February . .	603·0	23·74	− 2·5	27·9	62	62	110	4·4	16	14	16	..	..	81·1	108·3
March . . .	604·5	23·79	− 5·4	22·3	62	60	93	3·7	16	15	16	..	..	123·9	98·3
April . . .	608·8	23·96	+ 0·3	32·5	66	66	92	3·7	12	11	12	2	..	121·9	149·6
May. . . .	609·1	23·98	+ 4·6	40·3	73	75	130	5·2	16	15	12	1	..	98·1	
June . . .	612·7	24·01	+ 9·8	49·6	66	63	80	3·2	10	10	1	1	2	121·4	
July. . . .	615·1	24·21	+12·2	54·0	71	54	170	6·8	16	15	2	..	6	174·0	
August . . .	613·4	24·14	+ 9·8	49·6	76	59	233	9·3	17	15	..	..	3	167·4	
September . .	616·4	24·26	+10·7	51·3	64	46	35	1·4	10	8	..	..	..	176·4	
October. . .	616·4	24·26	+ 4·8	40·6	57	39	37	1·5	8	8	3	1	..	156·5	
November . .	609·0	23·97	+ 0·3	32·5	64	52	64	2·6	11	9	10	..	..	71·2	
December . .	614·7	24·19	− 0·9	30·4	51	42	69	2·8	9	7	9	..	..	88·3	

Digitized by Google

II.—Meteorological Table.—Comparison between Zürich and Arosa.

1898.

	Sunshine in Hours.		Winds.																
			Zürich.									Arosa.							
	Zürich.	Arosa.	N.	N.E.	E.	S.E.	S.	S.W.	W.	N.W.	Calm.	N.	N.E.	E.	S.E.	S.	S.W.	W.	N.W.	Calm.
January . . .	39·0	146·5	7	7	1	3	1	3	1	4	66	..	1	2	1	2	2	33	5	47
February . . .	80·3	76·5	3	2	5	1	6	15	21	5	26	..	1	11	4	2	..	9	1	56
March	93·7	132·7	4	9	9	1	2	12	9	7	40	..	..	4	11	..	4	16	1	57
April	142·7	140·2	7	13	6	1	3	5	13	8	34	..	..	10	3	1	9	12	..	55
May	136·1	150·9	4	9	2	2	6	14	15	17	34	..	..	10	11	4	13	3	..	52
June	169·8	130·4	2	6	7	5	11	7	13	3	36	..	1	13	5	6	1	2	..	62
July	238·0	199·6	5	10	3	1	6	12	9	11	36	..	2	25	5	3	..	1	..	57
August	304·6	275·1	2	5	5	3	2	5	10	6	55	..	..	19	10	2	2	..	..	60
September . . .	242·6	240·7	4	17	5	5	2	3	8	8	38	1	1	15	13	2	2	2	..	54
October . . .	70·3	138·3	2	9	5	4	6	6	11	9	41	1	1	22	5	3	5	1	4	51
November . . .	87·8	97·0	8	11	9	7	2	3	7	4	39	1	1	9	7	1	12	10	2	47
December . . .	50·6	119·3	3	3	6	2	9	8	12	3	46	..	1	4	..	..	5	20	2	61
The whole year .	1595·5	1847·2	51	101	63	35	56	93	129	86	491	3	9	144	75	26	55	109	15	649

Digitized by Google

disagreeable and weakening night sweats, whilst the fever continually diminishes, and finally disappears. It is to be remembered that feverish patients, even while lying in bed, are always in contact with the excellent air entering into their rooms through the windows and doorways, which are always kept open.

In some cases the cough and expectoration are increased on arrival, but they soon become less troublesome, and enable the patient to obtain a good uninterrupted sleep.

Examination of the lungs shows a progressive diminution of the râles, a drying up of the affected parts, and a continual improvement in the process of healing.

Finally, the psychical influence of the surroundings counts for much. The glorious sunshine and the grandeur of the mountain scenery infuse new hope and courage into the patient. I remember that it was one of the most memorable moments of my life when I, who had myself come as patient to Arosa, began to feel again the pulse of recovery and health.

About the Contra-indications.—We acknowledge that the high-mountain cure is contra-indicated in cases of—

Organic heart diseases.
Diseases of the kidneys.
Diabetes.
Tuberculosis of the intestine (peritoneal tuberculosis).
Psychical abnormalities.
Grave affections of the larynx.
Grave anæmia.
Extensive cases of tuberculosis of the lungs, with cavities.
Hectic fever and an incurable tendency to deterioration.

Fever in early cases need not be considered as a contra-indication; I have already mentioned how such fever lessens and disappears sooner in the pure mountain air than in the lowlands.

Inclination to hæmorrhages, moreover, is not a contra-indication.

Out of 120 patients of mine at Arosa during the last winter season, only 12, that is, 10 per cent., had hæmorrhage while at Arosa.

Fifty of the 120, that is, 41·6 per cent., had previously suffered from hæmorrhage in the lowlands, and of this fifty only *ten*, that is, 20 per cent., had a recurrence of hæmorrhage while in the mountains.

Two cases out of the 120, that is, 1·6 per cent., suffered from hæmorrhage only at Arosa and not before.

From the consideration I have set out in this paper I am convinced that the high-mountain cure, carried out under careful medical supervision, remains the best that can be given to people affected with tuberculosis of the lungs.

Such persons (provided they are free from the contra-indications before referred to) may be sent *at once* to the high-mountain cure resorts at *any time*.

Digitized by Google

The chief climatic therapeutic agents are *always* there, and the researches of Dr. Turban, of Davos, have shown that the time of snow-melting—contrary to the opinion formerly received—has not any harmful influence on tuberculosis cases.

The mountain cure has great advantages over the cures made on the Riviera or similar places in two respects. Firstly, that the mountain cure can be effected at any time of the year, and is to a great extent independent of the weather; and, secondly, that those cured in the high mountains are much better able to bear the rigours of a lowland winter, and are not, like the patients from the south, obliged every winter to return to their place of cure.

Lastly, I should like to refer quite shortly to the influence of a high mountain climate on cases of

Surgical Tuberculosis.

Wölfflin, of Bâle, in collaboration with Bernhard, of Samaden, in the Engadine, found that surgical tuberculosis showed a quicker and better process of healing *post operationem* in the high-mountain districts than it did in the lowlands, and that the conservative method of surgery has a better chance. Especially he saw astonishing influence on chronic lymphomata, on tuberculous affections of the articulations and of the bones, of the uro-genital apparatus, and probably on lupus.

I myself obtained a very good result in seven cases of multiple lymphomata disappearing or diminishing without any operation except lancing, and a beautiful healing after the operation of scraping out a caries of the ninth rib.

With Wölfflin and others I strongly recommend a period of residence at a high altitude as a prophylactic to young people, who are now sent more frequently to these high-mountain places since the fear of infection has been vanquished by the existence of sanitary and disinfection laws in these high-mountain health resorts.

I could give you only a short account of what there is to say about the high-altitude treatment and its principles, but I shall be very pleased to answer any question which may be asked on the subject of this paper, and it only remains for me to express my thanks to you for the attentive way in which you have been pleased to listen to my paper.

Digitized by Google

TUBERCULOSIS IN THE MALTESE ISLANDS.

By Dr. THEM. ZÀMMIT, *Malta.*

The mortality from tubercle in Malta is exceptionally low. As the general death-rate is not very low, and as overcrowding and the other debilitating agencies are by no means rare, the comparative freedom from tubercle must be due to the sum total of the telluric and atmospheric conditions which constitute climate.

It is a well-known fact in the islands that tubercular diseases in the cattle and among the domestic animals are of rare occurrence.

The percentage of deaths from tubercle in relation to the general death-rate is as follows:—

During 1890 . .	9·2	During 1895 . .	6·8
„ 1891 . .	9·3	„ 1896 . .	8·6
„ 1892 . .	8·9	„ 1897 . .	8·1
„ 1893 . .	9·2	„ 1898 . .	10·7
„ 1894 . .	6·3	„ 1899 . .	9·4

The average being 8·65 for ten years.

Comparing this with the proportion of deaths from tubercle to the general mortality of other countries, we find that Malta stands at the bottom of the list.

Detailed tables gathered from the official reports of the Public Health Department of Malta are given to demonstrate the fact.

PERSONAL EXPERIENCES IN CHEST-MEASURING IN CASES OF PULMONARY TUBERCULOSIS TREATED AT HIGH ALTITUDES.

By the late Dr. CARL RUEDI, *Arosa.*

Already in 1889 I tried to make chest diagrams of my patients. However, I did not carry out the idea as far as I should have liked to have done, and it was only last winter in Arosa that I took it up again and on a larger scale. I made at stated times diagrams, similar to those which I hereby send, of most of my patients, measuring them three times during the winter season, with a lapse of about eight weeks between each measurement.

The method I employed in measuring my patients and in drawing these diagrams was the following:—

With the calipers I measured the diameter of the chest from the *processus spinosus VIII.* to the junction of the *processus xiphoid* with the *sternum.* This diameter was taken when the patient breathed normally, and, as a rule, it was found that measurements taken either at the moment of inspiration or of expiration differed only in about three millimetres.

Next I used the tape, measuring the left and the right side of the chest

Digitized by Google

separately, and asking the patient not to breathe deeper than usual. Then I found the circumference of the whole chest, one inch above the nipple—the patient having his arms stretched out sideways—and noted the power of expansion the lung had.

Thirdly, I applied the cyrtometer—a rod of soft pliable lead—and determined the shape of the right and of the left lungs, the patient's arms hanging down at his side. Transposing the cyrtometer to a paper where the diameter obtained by the calipers had been marked off, I immediately had my diagram. The above-mentioned cyrtometer can easily be adapted to the body, and as easily brought back to its normal form, so that the diagrams thus obtained are accurate and of great use when comparing them.

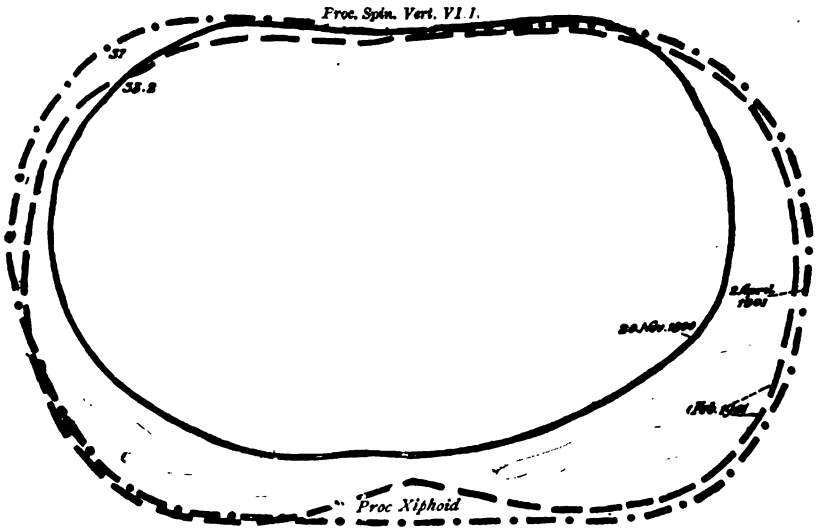
My experiences with this method have been very interesting, and it would certainly be advantageous for the specialists in chest diseases to become acquainted with the above method. It is only by means of these mechanical observations that new points for the diagnosis and prognosis of these diseases can be gathered. Most of the medical world thinks that the diseased part of the lung is badly developed or contracted, and it is this deformed side which is generally recognised as containing the diseased lung.

This is absolutely untrue, and I can prove by my diagrams that the unsound side of the chest in an early stage is always fuller and better developed than the opposite side, because the deposit of pulmonary inflammation in the lung tissue itself is resistant enough to hinder the chest-walls from being contracted. The deformed side of the chest in the so-called *first stage* of tuberculosis shows with the calipers larger diameters than the sound one. It is only when the disease has gone over into the second stage, and when in the connective tissue the infiltration becomes contracted and forms the cicatrisation known as cirrhosis of the lung, that the circumference of the formerly diseased lung becomes smaller. This is exactly as in pleurisy and empyema, where, as long as the disease is active, we always find an enlargement of the attacked side; and it is only later on, when the process has become stationary and a consolidation of the fibroid deposits has taken place, that the diameter and circumference of this side diminish.

The third stage is again quite different, and here it is that high altitude shows its most exquisite power. When the lung is once free from active mischief, and contraction of the diseased parts has set in, then the high altitude is the best regenerator of the lost lung tissue. In all of my examinations at Arosa I have found that the diminished atmospheric pressure has the tendency in every respect to develop the chest symmetrically—where there was infiltration a piece had to be contracted, where there was cicatrisation or cirrhosis there the lung had to expand, and the spare *alveoli* had to do their duty. The surrounding sound lung tissue had to expand and to fill out the places which had been deprived of their contents by cavities, etc.

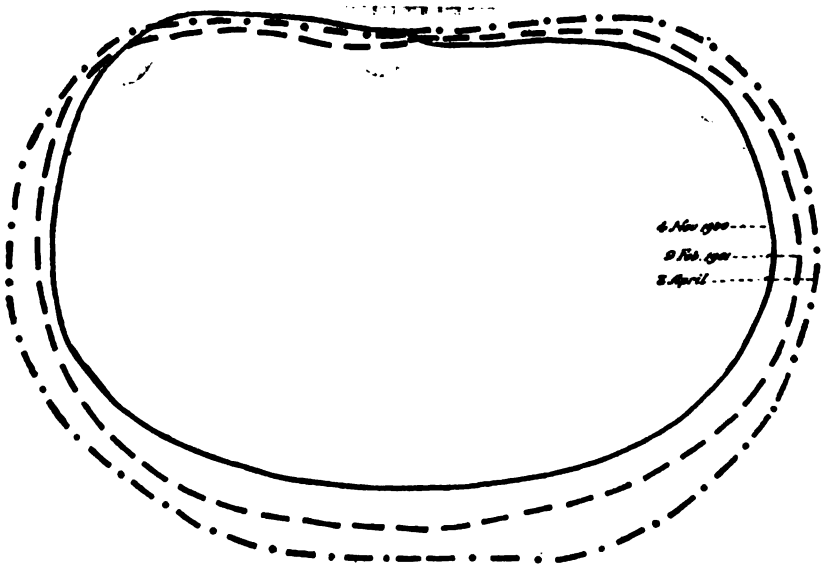
There were particularly two facts that always struck me—namely, that in high altitudes the circumference around the chest increases largely, and secondly, that this development of the chest is, without any exception that I could state, *symmetrical*.

Digitized by Google



I.

	Proc. Xiph. to Proc. Spin. VIII.	Circumference. Left.	Right.	Weight. Kilos.
November 20, 1900	13·8	33·5	37	50
February 1, 1901	14·6	37·2	38·2	50½
April 2, 1901	14·5–17·2	36	38	



I I.

	Proc. Xiph to Proc. Spin. VIII.	Circumference. Left.	Right.
November 4, 1900	17 to 20·5	38	39·5
February 9, 1901	17·5 to 21·2	40·2	41·6
April 8, 1901	19 to 22·5	43·5	43·2

Measures in centimetres.

Digitized by Google

STATISTIQUES ET RÉSULTATS DE LA CURE D'ALTITUDE DANS LA TUBERCULOSE PULMONAIRE.

PAR M. LE DR. TH. STEPHANI, *Beauregard, Montana, Valais.*

DE l'observation de 150 tuberculeux traités à Montana de 1897 à 1900 les résultats statistiques sont les suivants :—

Au point de vue des formes de la tuberculose :

34 % d'infiltrations sèches.
30 % de formes bronchitiques.
17 % de formes cavitaires.
13 % de formes congestives.
3 % de formes pleurétiques.
2 % de formes abdominales.
1 % d'adénites tuberculeuses.

Les lésions pulmonaires sont le plus souvent bilatérales (53 %) ou siègent plus fréquemment à droite (27 %) qu'à gauche (20 %).

Les complications laryngées entrent en ligne de compte 15 %.

L'hérédité existe moins souvent (44 %) qu'elle ne fait défaut (56 %).

La présence du bacille de Koch est démontrée dans les trois quarts des cas, soit dans la presque totalité des formes où l'expectoration a pu être recueillie.

La transmission de la tuberculose d'homme à homme est manifeste dans 80 % des cas, ce qui prouve l'importance de la contagion. Notre enquête démontre à ce sujet combien les casernes, les convents, les bureaux d'administration sont des centres fréquents de propagation tuberculeuse.

Le rôle initial joué par la plèvre est assez frappant, car dans 14 % des cas la tuberculose pulmonaire débute par une pleurésie et dans 18 % on trouve des accidents pleurétiques signalés plusieurs années avant l'éclosion de la phtisie.

Pour l'appétit, 97 %, la cure alpine donne une réelle amélioration qui se traduit 67 % par l'augmentation progressive du poids.

L'extension de la capacité thoracique, due surtout à la raréfication de l'air, est notée fort souvent.

Les troubles cardiaques imputables à l'altitude n'ont été remarqués qu'une fois sur 150 ; il s'agissait de tuberculose congestive avec vice de cœur déjà diagnostiqué en plaine.

Les troubles nerveux n'ont pas été observés.

Les haemoptisies ne se montrent pas plus fréquentes à l'altitude qu'en plaine, puisque 35 % des malades en avaient présenté chez eux et n'en ont pas eu au sanatorium et que 1½ % en ont eu pour la première fois à Montana.

Digitized by Google

La fièvre est souvent combattu très efficacement par le climat alpin ; elle a cédé en effet chez les 60 % de nos fébricitants.

Quant aux résultats au point de vue de la guérison même, la cure aérothérapique d'altitude donne :

12 % de guérison complète.
50 % d'amélioration et de demi-guérison.
20 % d'état stationnaire.
18 % d'aggravation et de décès.

100

Ce 12 % de guérison appartient aux formes suivantes :

Cavitaires fébricitants avec laryngite		0·7 %	soit le 4 % des cavitaires.
Congestifs apyrétiques . . .		0·7 %	„ „ 5 % des congestifs.
Bronchitiques	fébricitants 0·7 % apyrétiques 1·3 %	2·0 %	„ „ 7 % des bronchitiques.
Infiltrations sèches	fébril eavec laryngite 0·7 % apyrétique avec laryngite . 0·7 % apyrétique sans laryngite . 7·0 %	8·4 %	„ „ 24 % des inf. sèches.
Pleurétiques apyrétiques		0·7 %	„ „ 25 % des pleurétiques.
(Tuberculoses abdominales . . .		0·7 %	„ „ 33 % des tub. abd.)

Ce sont donc les infiltrations sèches (catégorie comprenant les $\frac{3}{4}$ des tuberculoses au début) qui fournissent le taux le plus élevé de guérison.

Les formes apyrétiques sont plus favorables que les fébriles. Les complications laryngées, bien que fâcheuses, ne rendent pas la guérison impossible.

Les $\frac{2}{3}$ des guéris étaient exempts d'hérédité, ce qui prouverait que les non-héréditaires ont plus de chances de guérison.

Conclusions.

1° La cure d'altitude est indiquée dans toutes les formes de la tuberculose.

2° Elle donne en bloc : 12 % de guérison complète.
50 % d'amélioration.
20 % d'arrêt dans le cours de la tuberculose.
18 % d'insuccès.

3° Les chances de guérison sont d'autant plus nombreuses qu'il s'agit d'une tuberculose au début, d'une forme pleurale ou sèche, apyrétique exempte de complications et d'hérédité.

4° Les seules contre-indications avérées de l'altitude sont avec les formes asthmatiques, les tuberculoses congestives accompagnées de troubles cardiaques.

Digitized by Google

STATISTICS AND RESULTS OF THE HIGH-ALTITUDE TREATMENT IN PULMONARY TUBERCULOSIS.

By Dr. TH. STEPHANI, *Beauregard, Montana, Valais.*

From observation of 150 tuberculous patients treated at Montana from 1897 to 1900 the following statistics are obtained :—

1. Form of the disease :

Dry infiltrations	= 34	per cent.
Bronchial types	= 30	„
Cases with cavitation	= 17	„
Congestive forms	= 13	„
Pleuritic cases	= 3	„
Abdominal forms	= 2	„
Tuberculous adenitis	= 1	„

The pulmonary lesions are more frequently bilateral (53 per cent.), and are more frequently situate on the right (27 per cent.) than on the left (20 per cent.).

Laryngeal complications rank in the proportion of 15 per cent. Heredity is present less frequently (44 per cent.) than it is absent (56 per cent.).

The bacillus of Koch was present in three-quarters of the cases, or in almost all those in which it was possible to collect the expectoration.

The transmission of tuberculosis from man to man was obvious in 80 per cent. of the cases, a fact which proves the significance of contagion. The inquiry we have made on this matter shows how many barracks, convents, and business offices are common foci for the propagation of tuberculosis.

The part played by the pleura as a starting-point of the disease is sufficiently striking, for in 14 per cent. of the cases pulmonary tuberculosis commences with a pleurisy, and in 18 per cent. one finds injuries to the pleura described as having taken place several years previous to the development of the phthisis.

2. As regards appetite, there is a decided improvement in 97 per cent. of the cases, which showed itself in 67 per cent. by a progressive rise in weight.

3. Extension of thoracic capacity, due above all to rarefaction of the air is frequently noted.

4. The cardiac troubles ascribed to high altitudes have only been noticed in one out of the 150 cases; it was a case of congestive tuberculosis, with malformation of the heart, that had already been diagnosed when in the plain.

5. Nervous affections have not been observed.

6. Hæmoptyses do not appear to be any more frequent at high altitudes than on the level, since 35 per cent. had manifested them when at home, and

Digitized by Google

not at the sanatorium; while only 1½ per cent. of the patients had hæmoptysis for the first time at the sanatorium.

7. Fever is often combated most effectually by the Alpine climate—it has yielded, in fact, in 60 per cent. of our cases.

8. As to the results from the point of view of actual recovery, high-altitude treatment gives the following:—

12 per cent. completely cured.
50 „ improved and half cured.
20 „ condition stationary.
18 „ condition worse, or died.

The 12 per cent. of cures may be subdivided thus:—

Febrile cases with cavities and laryngitis, 0·7 per cent., or 4 per cent. of cavities.

Congestive cases without fever, 0·7 per cent., or 5 per cent. of congestive cases.

Bronchitic cases with fever, 0·7 per cent.
„ cases without fever, 1·3 „
} 2 per cent., or 7 per cent. of bronchitic cases.

Cases of dry infiltration.
{ febrile with laryngitis, 0·7 per cent.
{ afebrile with „ 0·7 „
{ afebrile without „ 7 „
} 8·4 per cent., or 24 per cent. cases of dry infiltration.

Pleuritic afebrile cases, 0·7 per cent., or 25 per cent. of pleuritic subjects.

Cases of abdominal tuberculosis, 0·7 per cent., or 33 per cent. of cases of abdominal tuberculosis.

It is therefore the cases of dry infiltration (a group which embraces ¼ of incipient cases of tuberculosis) that furnish the most hopeful percentage of recovery.

Afebrile forms are more favourable than the febrile. Laryngeal complications, although troublesome, do not render cure impossible.

Two-thirds of the cured cases were free from hereditary taint, which goes to prove that the non-hereditary cases have a better chance of recovery.

Conclusions:

1. The high-altitude treatment is indicated in all forms of tuberculosis.
2. The treatment yields on an average:—

12 per cent. of complete cures.
50 per cent. of improvements.
20 per cent. of cases in which the tuberculous process is arrested.
18 per cent. of failures.

3. The more a case conforms to the type of an incipient tuberculosis of the pleuritic or dry form, free from pyrexia or other complication, and devoid of hereditary taint, the greater is the chance of recovery.

4. The treatment is only contra-indicated in cases of the asthmatic or congestive type accompanied by cardiac disease.

Digitized by Google

INFLUENCE DU CLIMAT SUR LA TUBERCULOSE OBSERVÉE DANS L'ARSENAL MARITIME DE BREST (FINISTÈRE).

PAR M. LE DR. C. AUFFRET,

Inspecteur-général du S^ce^ de Santé Ins^le^ de la Marine ;
Correspond^t^ de l'Académie de Médecine.

LA tuberculose pulmonaire est commune au port de Brest, et cette fréquence paraît être encore plus grande depuis quelques anneés. Sur 200 décès par an, à l'hôpital maritime, il y en a la moitié par cette maladie.

Il était de notre devoir, comme directeur du S^ce^ hospitalier qui nous est confié, d'en rechercher les causes et les moyens d'y porter remède. Nous l'avons fait dans un mémoire qui a paru l'an dernier et dont nous déposons quelques exemplaires sur le bureau du congrès.

Nous y avons suivi pendant trente ans un personnel de 7000 à 8000 ouvriers—nous garantissons l'exactitude de tous les chiffres. Nos recherches réunissent donc l'importance du nombre à la durée du temps, deux conditions que réclame toute importante statistique.

Nous nous bornerons à parler ici de l'influence du climat.

Il est démontré, depuis les travaux des illustres savants Villemin et Koch, que la contagion doit être inscrite en tête de l'étiologie et de la propagation de la phtisie pulmonaire ; mais on ne saurait nier l'action des causes qui y prédisposent et qui l'entretiennent, au 1^er^ rang desquelles se placent, comme le disait Verneuil, " toutes les affections qui font tousser." Or, le climat de Brest dispose aux bronchites et aux laryngites ; et non seulement il ouvre ainsi la porte à la contagion, mais il agit sur les rechutes et sur les recrudescences quand l'ennemi est entré dans la place.

C'est ce que nous constatons tous les jours.

Climat de Brest.—Le port de Brest est situé à l'extrémité d'une pointe avancée, comme l'avant d'un navire, dans la mer. Or J. Rochard a jadis établi l'influence nocive de l'air marin sur la marche de la phtisie pulmonaire.

D'une manière générale, le climat de Brest est constant, l'un des plus constants de France, d'après les recherches de Borius ; mais les variations journalières en sont marquées et fréquentes.

L'uniformité relative de la température est due au voisinage du Gulf-Stream, calorifère naturel qui en relève la température de 3°. Mais l'atmosphère, sous son influence, l'imprègne d'une buée épaisse qu'évaporent, pendant l'été, les rayons du soleil, mais que la température fraîche de l'hiver n'arrive pas à dissiper, qui imbibe les vêtements, et qui souvent retombe sous la forme d'une brume pénétrante, mouillant à l'égal d'une pluie fine et dense.

Digitized by Google

Si l'on consulte le baromètre, ces jours devraient être de belles journées ; et, somme toute, malgré leur extrême humidité, elles ne seraient pas mauvaises au point de vue des maladies de poitrine, si elles n'étaient si souvent modifiées en un instant, par des sautes de vent qui, du sud-ouest ou de l'ouest, passent brusquement au nord-est et à l'est.

Température.—La température de Brest est, en moyenne, de C. 11° à 11°7.

Elle augmente jusqu'à 3 heures du soir, tombe ensuite plus rapidement qu'elle n'était montée, les chutes en étant plus sensibles en été qu'en hiver, le minimum de la température précédant le lever du soleil ; peu de gelées ; mais quand les oscillations se font dans un court espace de temps, elles sont d'autant plus senties que les températures moyennes sont accompagnées d'une humidité pénétrante.

Vents régnants.—Les vents les plus fréquents, et dans une très grande proportion, sont le sud-ouest et l'ouest ; alternant avec les vents de nord et de nord-est. Les premiers sont les vents humides et tièdes ; les seconds les vents secs et frais. Ce sont ces derniers, qui répondent à un ciel sans nuages, à ce que l'on est convenu d'appeler un très beau temps, qui causent l'abaissement brusque de la température pendant l'hiver et le printemps.

Ce sont les transitions *rapides* qui occasionnent journellement les affections aiguës des voies respiratoires, irritant la muqueuse des voies, et ne cédant qu'au retour du vent chaud et humide.

Ces retours des vents secs et frais agissent cruellement sur les phtisiques, qui sous leur influence, surtout dans la saison d'hiver et les premiers mois du printemps, reviennent prendre un lit d'hôpital, et l'on constate chez eux des poussées d'autant plus longues que la période de vent de nord-est a été plus tenace.

Pluies.—Ch. *Guépratte*, directeur de l'observatoire maritime, a observé pendant plus de 30 ans avec un pluviomètre rectangulaire (m.c.) une moyenne de 986 mill. ; *Belleville*, après lui, en 6 ans a obtenu 730 mill. ; *Kermarec*, en 8 ans, 755 mill.

Le climat de Brest est donc très pluvieux, et paraît l'avoir été un peu plus dans la première moitié du siècle.

Il est entendu que le climat n'engendre pas la tuberculose.

Pour bien en établir la nature contagieuse nous avons fait analyser les poussières de tous les ateliers de l'arsenal. Nous y avons trouvé le bacille de Koch, spécialement dans tous les ateliers ayant des planchers à rainures.

Donc l'élément contagieux, le contage, y existe ; mais quand la tuberculose est déclarée, les variations de température brusques l'exaspèrent et l'aggravent, et s'il est vrai, ce qu'il faudrait démontrer, que l'air uniformément sec et frais est un bienfait pour les phtisiques, nous pouvons affirmer après de longues observations, que l'alternance d'une humidité tiède et d'un froid sec leur est funeste.

Rien ne vaut, pour établir l'influence climatérique, la comparaison entre la courbe indiquant les moyennes de la température et celle qui indique le nombre des décès pour chaque mois dûs à la tuberculose.

Borius avait déjà remarqué qu'il y a un retard du mouvement des décès

Digitized by Google

sur la température et qu'il faudrait avancer d'un mois sur la gauche la courbe des décès pour que les deux se confondissent presque en une seule. On constate alors que plus le temps est froid plus les décès par phtisie pulmonaire sont nombreux, et ils atteignent leur chiffre minimum dans le mois qui suit la température maximum.

Les décès mensuels, par les températures extrêmes, sont donc en raison inverse des mois qui les précèdent immédiatement.

Ces faits peuvent être constatés dans la courbe que nous présentons (30 ans d'observation). *Voir le mémoire.*

TUBERCULOSE.—INFLUENCE DES SAISONS.

Courbe des décès par mois de 1869 à 1898 (6500 à 7500 ouvriers).

Janvier.	Février.	Mars.	Avril.	Mai.	Juin.	Juillet.	Août.	Septembre.	Octobre.	Novembre.	Décembre.
96	112	120	121	90	54	56	50	52	78	76	89

INFLUENCE OF CLIMATE—THE CLIMATE OF BREST IN PARTICULAR.

BY DR. AUFFRET, *Brest (Delegate of Marine Ministry).*

THE relatively temperate climate of Brest ought to be favourable to patients suffering from pulmonary tuberculosis, but it is not.

First of all sea air is usually harmful.

But most harmful of all are the sudden atmospheric changes, almost instantaneous transitions from a mild humid temperature (west and south-west winds) from the warming Gulf stream, to a dry, cold temperature (north and north-east winds).

The following are our conclusions:—

1. Mortality from pulmonary phthisis is in inverse ratio to the mean temperature of the month which precedes the deaths.

2. Variations of temperature undoubtedly influence the progress of phthisis and conduce to precipitate (hasten) death, but that is all.

3. The most trying months are those from December to April, March being the worst of all. These months inherit the harmful influence of winter.

Our most recent statistics are absolutely in accordance with these conclusions.

We shall present to the Congress in London statistics representing the influence of climate on mortality during the last thirty years.

Digitized by Google

WEDNESDAY, JULY 24th.

COMBINED DISCUSSION WITH THE SECTION OF PATHOLOGY ON THE THERAPEUTIC AND DIAGNOSTIC VALUE OF TUBERCULIN IN HUMAN TUBERCULOSIS.

G. A. HERON, M.D.,

Physician City of London Hospital for Chest Diseases.

In the autumn of 1890—all but eleven years ago—there took place in Berlin one of the most remarkable events that mark epochs in the history of medical science. At that time, in the beautiful capital of the German Empire, there were gathered together some thousands of practitioners of medicine. Among them were many men of the highest rank in their profession, men whose names and whose works were well known to all students of medical literature. Great centres of learning, famous scientific societies, medical schools and colleges, sent their representatives. Both hemispheres, and all countries of importance, were represented at this memorable gathering. The reason why the medical profession then flocked to Berlin was not because a new method of treating tuberculosis had been announced, but because a new method of treating that disease had been made known, as the work of his own hands, by the man who shared with Lister and Pasteur the honour of having done most for the advancement of scientific medicine, and the consequent lessening of human suffering. The great gathering in Berlin in 1890 was but an expression of the admiration and respect in which Robert Koch was held; for it is certain, that had tuberculin been first brought to our knowledge by a man not in the first rank of our profession, the remedy would have been left to find its own way slowly—perhaps very slowly indeed—into notice.

With much enthusiasm, and with high hopes of its future usefulness, tuberculin was received in the autumn of 1890. Before the end of the following spring, the remedy had, to a very great extent, fallen into disuse, and was damned as loudly as it had ever been welcomed. The outcry against the use of tuberculin—for it was nothing less than this—had for its chief cause the condemnation of its use by another distinguished German, Virchow. The patriarch of present-day pathologists said that, in his opinion, the use of

Digitized by Google

tuberculin was fraught with danger to the patient. His reason for making this statement was that he had seen in the bodies of consumptives, who in life had been treated with the old tuberculin, evidence which convinced him that the drug caused destruction of the tissues around tubercular centres, and so set free the bacillus to do its work upon healthy tissues. Virchow maintained that, had the remedy not been used, the bacillus might have rested, harmless, encapsuled, as we know it often is, by tissues that have undergone an indurative process, and become fibrous. Now this statement of his views by a man so justly famous as Virchow at once attracted attention. There is, I think, no doubt that this expression of opinion greatly tended to deter men from using tuberculin, and to prejudice the medical profession everywhere against its use. And so tuberculin was both made and unmade in Germany!

Of Virchow's objections to the employment of tuberculin I can only say that, in my hospital experience of its action, there has been observed no evidence of resulting spread of disease to seemingly healthy organs, or to neighbouring tissues. The average stay in hospital of the first thirty-five cases treated with old tuberculin by me extended to sixty-one days. There was, therefore, sufficient time in which to observe whether or not, in these cases, there was to be obtained clinical evidence in support of Virchow's views. In no case was this evidence found, and it was often searched for by other physicians besides those officially present in the wards.

It may be worth while to refer, in this connexion, to a case treated by me with the new tuberculin in hospital, and selected for that treatment because the patient was certainly slowly dying of extensive and advanced tuberculosis of both lungs. Dr. Perkins, who is one of the Honorary Secretaries of this Section of the Congress, and was then pathologist to the City of London Hospital, performed the post-mortem examination. He made special search for the presence in the viscera of centres of probable fresh tubercular infection, but found none. This patient received twenty-six injections of new tuberculin in forty-two days, and the doses ran from $\frac{1}{100}$ mg. up to 2·5 mg.

Koch had, in the plainest words, and on several occasions, stated that cure of tuberculosis, when it affected the lungs, could be reasonably looked for, with the help of tuberculin, exclusively in those cases where only a small portion of one lung was infected to a slight extent, and where, of course, there was no evidence of excavation of lung tissue. He also said it would be reasonable to hope for a cure when only a small portion of each lung was diseased, there being no evidence of excavation. Any departure from these types of cases, in the direction of increase of the extent of the disease, lessened by so much, in Koch's opinion, the chances of a cure; although even in more advanced cases amelioration of symptoms was, he believed, likely to result from the use of the remedy. Now, hospital experience taught me that it is seldom we have there an opportunity of treating these early cases. Therefore it is not common to find in hospital the cases in which cure of the disease can be reasonably expected. So far as my experience of private practice goes, I must say the majority of cases in which I have been asked to advise as to the use of tuberculin have been too far advanced in

Digitized by Google

tuberculosis to permit of high hopes for the best result of the treatment being obtainable. But when in unsuitable cases the use of tuberculin produces no good result, blame should not rest on the remedy. In my opinion, tuberculin has fallen into discredit:

1. By its frequent use in unsuitable cases.
2. By its administration in too large doses.
3. By neglect of the rule that a dose of it should never be given until the patient's temperature has been normal for the previous twenty-four hours at least.
4. By neglect of the rule that the dose of tuberculin should never be increased, but, on the contrary, should be diminished, when its administration has been followed by a rise of temperature.
5. By the prejudice raised against the remedy, among both doctors and patients, because of the severity of the symptoms which not seldom follow upon its use.

Sir, I am obliged to crave indulgence of this meeting. It has been, if I may be allowed to say so, a very bitter disappointment to me that in consequence of a long illness, which, until a fortnight ago, unfitted me for work, I was obliged to relinquish the treatment of the cases I had begun to observe for the purposes of this discussion. Therefore, I am unable to present to you the new work which it was my earnest wish and intention to have submitted to you to-day, and I can deal only with work I have already published.

Since the end of 1890, fifty-seven cases have been treated with tuberculin by me at the City of London Hospital for Diseases of the Chest. Fifty-one of these were examples of tuberculosis of the lungs and six of lupus vulgaris. Of these, five of the lupus cases and twenty-seven of the others were treated with old tuberculin.

One case of lupus, and twenty-four cases of tuberculosis of the lung, were treated with the new tuberculin.

Seventeen of the fifty-one cases of lung-disease were lost sight of, practically from the time they left hospital. Sixteen of the remaining thirty-four were then known to be well and earning their living. I ask you to bear with me while I touch upon a few of the more important features—as I deem them—of these cases.

Whether consumptives leave a hospital well or ill is a very small matter compared with the answer to the really important question: How many of these people are able to work for their living, and for how long a time did they continue to gain their livelihood? I think the return to useful life of sixteen out of thirty-four cases of tuberculosis of the lungs, is not an unsatisfactory result of treatment, and this result was obtained with the help of tuberculin. According to our information at the hospital, ten of these sixteen cases of recovery are known to have remained well, and able to work for seven years, three for over three years, and three for nearly two years. Other patients remained able to work for periods varying from a few months to eighteen months; but as they are known to have broken down in health they are not included among these sixteen cases of apparent recovery.

Digitized by Google

Thirty-two of these fifty-one cases left hospital in 1891. Seven years afterwards—in June, 1898—the information concerning these cases, obtained, for the most part, by the assistant medical staff and nurses of the hospital, was as follows:

Eight of them were dead;
Ten were well;
One relapsed in 1897, having remained well until the autumn of that year;
Thirteen were lost sight of very soon after they left hospital.

These patients were all treated by the old tuberculin, and their cases are dealt with in some detail in a paper I contributed to Vol. XIV. of the *Transactions of the Medical Society of London.*

In addition to these thirty-two cases of tuberculosis of the lungs, five cases of lupus vulgaris were then treated by the old tuberculin. These cases all did remarkably well up to a certain point, and then, at longer or at shorter intervals, suffered relapse. In one of the five a relapse did not set in until more than a year after she left hospital, and she would not return for treatment because she said she could not afford to give the time necessary for this purpose.

Some of these cases of lupus vulgaris relapsed while under the influence of tuberculin. It is certain that at this period of the history of tuberculin, cases of lupus received unnecessarily high doses of the drug. When relapse took place in, for example, three of these cases, one of them had gained 10 lbs. in weight, and was taking 400 mg. doses of tuberculin; the second was taking 1000 mg. doses, and had gained 10 lbs. in weight; the third had gained 18½ lbs. in weight and was taking doses of 1000 mg. These large doses were given once in a week or a fortnight, and were in these cases not followed by high temperature, nor by other symptoms due to the action of the drug. The patients had been respectively 124, 128, and 135 days in hospital, and had received in that time, the first-named 50, and the other two 59 hypodermic injections of old tuberculin. It seems to me difficult to believe that these large doses could have exercised any material influence in the direction of favouring any tendency to relapse. The evils said to result from tuberculin are ascribed to its violent effects upon the tissues, by which, during severe reactions, infections of neighbouring healthy tissue is, according to Virchow's observations, apt to happen. But in these cases there were certainly no reactions when large doses of tuberculin were given, beyond slight redness round the site of the lupoid patches. There were no headaches, no rigors, no high temperatures; and yet, in these circumstances, relapse did take place, and while the drug was being administered. If the tubercle bacilli were let loose into the tissues, near the patches of lupus, as a result of severe reactions early in the treatment of these patients, then the drug should, one may reasonably urge, have had ample opportunity to exercise a beneficial influence, owing to the administration of the very large doses, which were not followed by reactions worth noticing. Certainly, in all but one of my lupus cases,

Digitized by Google

tuberculin seemed, after a certain time, to lose its power for good. I confess I have never been able to understand why, in cases which had improved so much, certainly because of treatment by tuberculin, relapse should have occurred while the treatment was in full use.

Since March, 1897, I have used only the new tuberculin. During that year ten cases were so treated in hospital. Two of these cases died. One of them was the man whose case I have already spoken of as having been selected because he was certainly dying. The other eight cases of this year were made up of seven examples of tuberculosis of the lung, and one of lupus vulgaris. They all did very well, and, without exception, left hospital, urging as their sole reason for leaving their fitness for work and their wish to resume work. In December, 1900, three years after treatment, the following was the result of the use of tuberculin in these ten cases :—

Two were dead, both of them recognised as being hopeless cases from the first ;
Three were well and supporting themselves by their work ;
Three were lost sight of ;
One remained well until lately, and returned to hospital a few weeks ago because of a recurrence of disease.

No. 10 of the patients of the year 1897 is worthy of some notice. It is the only case of lupus I have treated with the new tuberculin, and it is the only case of that disease, which in my hands, has not suffered relapse. He was a coachman, 27 years of age, admitted to hospital on April 17, 1897, with lupus vulgaris affecting the whole of his left nostril, both within and without; about one-half of the anterior part of the right nostril, within and without; and the bridge of the nose between the alæ nasi, excepting the tip of the nose. The left lip close to the middle line, at junction of lip and ala nasi, was also diseased. There was another infected spot of skin three-quarters of an inch in front of the lobe of the right ear. This patch was about the size of a sixpence. Treatment with new tuberculin was commenced April 20, 1897, and completed August 4, 1897. Total quantity of tuberculin injected (hypodermically) 126·5 mg., given in sixty-three injections ; the doses ranged from $\frac{1}{5000}$th part of a mg. to 4 mg. On July 2 scar tissue only was seen over sites of lupus patches, excepting the patch on the left lip; this had entirely healed sixteen days earlier. On February 25, 1898, I showed this patient to the Clinical Society of London. He was then free from recurrence of his disease. To complete this part of the case I may say he was quite well on June 9, 1898.

In the discussion which followed upon the exhibition of this case at the Clinical Society, it was asserted that the diseased condition I have described to you was due to syphilis, and not to lupus. In my notes laid with the case before the society, it was stated that the man had a history of gonorrhœa followed by sore throat. On his shins were two or three brownish stains, which, together with the history just mentioned, made it not improbable he had had syphilis. He said he had never had skin disease until the appearance of

Digitized by Google

the lupus patches on his nose. They began, he said, with a pimple. You will observe in this connection that it was stated in the hospital notes of the case, that there was on July 2 only scar tissue over the sites of the principal lupoid patches. On July 5 this patient, in going about the hospital, hit his shin sharply upon the angle of a chair. The skin was bruised and cut, and the patient suffered considerable pain in consequence. When, a day or two after the accident, I saw the shin, it was very tender, and the man complained of pain. I ordered him to have gr. 5 doses of iodide of potassium, and this treatment was continued for a week. He was then free from all uneasiness, and the swelling was gone. The use of the iodide was then permanently discontinued.

It seems to me that it can hardly be fairly argued that this episode of the iodide had any effect upon the skin disease that had affected the nostrils, but practically was cured before the iodide of potassium was first given. Another point about the case is, that the patient's temperature, taken for a week before the first dose of tuberculin was given, was normal. After a dose of $\frac{1}{300}$th of a mg. of tuberculin, his temperature rose in twenty-four hours to 100·2°, and after a dose of $\frac{1}{100}$th of a mg. to 100°. This patient's temperature charts show that he was peculiarly susceptible to the influence of tuberculin. Doses of 1 mg. caused temperatures of over 102 on three different occasions, and $\frac{1}{2}$ths of a mg. caused similar temperatures to be recorded. During and after a rise of temperature, the characteristic halo of red skin was seen around the diseased parts. To my mind these facts are most important in helping us to a diagnosis of this case.

At this discussion of the Clinical Society of London, one dermatologist of note and a well-known laryngologist were emphatic in their statements as to this case being one of syphilis. I hope I shall not be blamed because I attach more importance to the behaviour of this patient's temperature and lupoid skin under the influence of tuberculin, than to the expressions of opinion to which I have referred. The laryngologist was particularly convinced as to the accuracy of his observations, and that the case was one of syphilis, because, he said, he smelt dead bone in the patient's breath, and saw evidence of dead bone on inspecting the patient's nasal cavities with his laryngoscope. Four months after that expression of laryngological opinion, the patient was quite well, and had shown no evidence of the presence of dead bone in his nose or elsewhere; but whether or not his breath smelt of dead bone I cannot tell, because my nasal acumen in that direction is obviously untrustworthy, for I could not, at any time, detect the odour of dead bone which was so plainly revealed to the no doubt specially educated olfactory system of the laryngologist.

In 1899, ten patients, five men and five women, were treated in the wards with the new tuberculin. Of these five women, two are known to be still in good health. One of the two is a domestic servant; the other is a shorthand writer and typist. Two other women are reported to have broken down in health. The fifth has not been heard of since she left the hospital.

Of the men, one continued well for more than a year after treatment, but appears to have recently had a relapse. He worked as a hawker, and so

Digitized by Google

supported himself. Another seems now to be as well as he was when he left the hospital, and is said to be able to work. The third died November 16, 1900; the cause of death was not stated to us, but he was carried from his work, and two months afterwards died in his bed. Probably he died from a relapse. The fourth was reported to be very ill a year ago. The fifth is working as a photographer's assistant, but he says he had spitting of blood two months ago.

In Krause's record of his six years' experience of Koch's method of treatment of tuberculosis, he states that of twenty-seven of these cases, twelve are well, thirteen improved in health, one *in statu quo*, one is worse. It is, of course, not possible on an occasion like this to go into details at any length; but some authors have more or less emphatically dissented from the outcry raised against the use of tuberculin—*e.g.*, Hansen, of Bergen, Osler, Gœtsch, McCall Anderson, Petruschky, Spengler, and others. Those of us who have dissociated ourselves from the outcry against tuberculin, have all expressed the view that in cases of tuberculosis, which fall within the limitation laid down in 1890 by Koch, great benefit to the patient is sure to follow upon a proper use of tuberculin, and very often there is complete disappearance of all symptoms of activity of the disease.

By far the most important communication, after Koch's own, that has yet been made to the literature that deals with tuberculin, was published by Dr. Gœtsch, of Slawentzitz, on May 1, 1901. If the patient can well bear the old tuberculin, Dr. Gœtsch conducts the whole treatment of the case with it, beginning with so small a dose as ·00001 grm. If this small dose should cause a rise of temperature, then Gœtsch goes back to the new tuberculin, giving it in doses of about ·001 mg. Having felt his way by carefully avoiding the use of doses which would at all raise the temperature, or produce other symptoms of reaction, Gœtsch substitutes in the treatment the old for the new tuberculin. Since 1891 Gœtsch has treated 224 cases of tuberculosis, not of the lung only, but of all tissues. Of these he says he has cured 71%.

The record of these cases is most interesting, and the reading will, I think, carry, to any unprejudiced mind, the conviction that Gœtsch has given us a very important new departure in the use of tuberculin.

Responsible opinion is unanimous in its recognition of the worse than uselessness of any attempt to treat with tuberculin, tuberculosis that is complicated with other inflammation. These mixed infections, as they are called, were not recognised in 1890; and it is only within the last few years that their great importance as complications of tuberculosis, whether of the lungs, of the skin, or of any other tissue, has been clearly apprehended.

Is there good ground for the belief that tuberculin, when properly used, is dangerous in any degree to the patient? From my own experience—and I am responsible for over 2000 injections of it—I am strongly of the opinion that it is, at least, as safe to the patient as is any other very potent drug.

Having said this, I will ask your attention to a statement of a case of great interest, which, in all its details, is well known to me.

A girl, seventeen years of age, was submitted to a test injection of

Digitized by Google

tuberculin, because her physician had a doubt as to whether or not the girl had tuberculosis. She was thin and under-sized. There was dull percussion over both lungs, and abundant crepitant râles filled them both from apices to bases. Her liver and spleen were enlarged. No tubercle bacilli were found in the sputum. When the question of giving this girl a test dose was discussed, it was decided that, lest her symptoms should be due to tuberculosis, only a minute dose of old tuberculin should be given. Therefore, instead of the usual initial test dose of 1 mg., one-third part of a mg. was injected in the usual way, and with, of course, strict attention to antiseptic precautions.

I show you an enlarged copy of this patient's temperature chart. The temperature was taken in the mouth, and the chart records it morning and evening for eleven days previous to the injection of the tuberculin. From the time of the injection onwards, the temperature was taken every four hours. Before the injection, the temperature, you will observe, ranged between two to four points below 98° up to a point or two above 99°. After the injection the temperature never rose above 98°, and it fell on the last day to 96·4° and 97°. Respiration rose gradually from about twenty to double that rate. The heart's action became feebler and feebler, until she died twenty-seven hours after the giving of the injection. There was vomiting two hours, and again three hours after the injection. For the last twenty-four hours of life the patient had diarrhœa, not bloody.

It happened that two other women and two men each received the usual test dose of 1 mg., taken from the same bottle that had been used in this fatal case. Both of the women had moderate reactions, but one of them became jaundiced, and had a considerable amount of vomiting. The other woman had a rise of temperature to 102°, and no other symptoms, excepting only a slight sense of headache. Both the men had severe reaction with rigors, and a rise of temperature in one case to 104·4°, in the other to 102°. The man whose temperature reached 104·4° had vomiting, which, at intervals, continued for five days.

I need not say, a series of cases like this demanded a most rigid investigation as to the cause of these deplorable results, plainly associated, as they were, with the injection of these test doses.

As regards the case ending in the death of the patient: that the death was not due to tuberculin seems beyond doubt, for dangerous symptoms have, so far as I know or can ascertain, never been observed to follow a dose of less than 1 mg. The characteristic of the action of tuberculin is a rise of temperature. Here the temperature fell. Vomiting is a not infrequent symptom of a severe reaction, but here there was no evidence of reaction due to tuberculin. Excepting only this case, I have no knowledge of diarrhœa having ever followed upon the use of tuberculin. But the symptoms of jaundice and vomiting in the other woman and the five days of occasional vomiting which occurred in the man, made it certain something was wrong with the injection used in these cases. It was contained in one of several bottles, all filled with tuberculin of the same date of manufacture; the date being one month and eleven

Digitized by Google

days previous to the giving of injections. All these bottles were sent to Dr. Macfadyean, of the Jenner Institute. They were unopened, excepting, of course, that one which had already been in use. He examined their contents by every method known to bacteriologists. He says in his report: "The tuberculin was injected into guinea-pigs in doses of 1 cub. c.; 0·5 c.c., and 0·2 c.c. No toxic effects were produced, nor any noticeable symptoms." Dr. Macfadyean ends his report with these words: "I have not, therefore, been able to trace any abnormal or deleterious property in connection with the samples of tuberculin. . . ."

The doses of the drug were made up by a most efficient druggist, and careful enquiry made it certain there could have been no error in the dosage. Of course every effort was made to secure a post-mortem in this case, but the necessary permission could not be obtained.

One may speculate upon the cause of this death; and it may be suggested, with much force of reason, that it was due to widespread and advanced degeneration of the intestinal glands. But the absence of post-mortem evidence, and the want of all help from the bacteriological examination, must, I fear, for the present, bring us face to face with a deadlock in seeking to explain this case.

The fact that this girl showed no reaction proves to the minds of, I think, all those who have much experience of the use of tuberculin as a test for the presence of tuberculosis in human and other animals, that it is highly improbable she had tuberculosis.

What, then, is the diagnostic value of tuberculin? That it produces its characteristic reaction wherever tuberculosis is present, there can be no doubt. That it rarely fails to react where there is tuberculosis is so true, that cases in which failure is recorded may safely be neglected. It produces no evil effects in such cases as disease of the larynx, nor in kidney disease with albuminuria and granular tube-casts, nor in disease of the bladder, nor, so far as I know, in any condition of disease, whether tubercular or not. As an illustration of the practical usefulness of tuberculin as a test in cases always difficult and often impossible of diagnosis by ordinary methods, I would refer to the excellent work done in this direction by Dr. Eric France, of the London County Asylum, Claybury. His object was to ascertain, with certainty, who among the insane inmates of the asylum had tuberculosis. For this purpose he tested fifty-five of his patients with tuberculin. Characteristic reactions occurred in forty-five of these cases. Thirty-four of them eventually died, and twenty-nine of these thirty-four were submitted to post-mortem examination, with the result that, as Dr. France says, "Active tubercle was found in every case." Ten of the fifty-five patients did not react. Five of those died, and post-mortem, says Dr. France, "No trace of tubercle found in any; five still alive and healthy." Here is his expression of opinion on this matter in his own words: "I injected seventy-five cases with tuberculin, and personally I am satisfied, not only with the accuracy of its diagnostic power, but also with its entire harmlessness, both in the tubercular and in the non-tubercular."

We know, sir, that the best, I had almost said the only, chance of cure of the consumptive is to treat him in the earliest days of his illness. It is a fact

Digitized by Google

that by no other means can we so early diagnose tuberculosis in men as we can by the help of tuberculin. It is also a fact that we can do this with safety to the patient. Had tuberculin served no other good purpose than this, it would have deserved our best attention, our high appreciation. I am sure the day is not distant when the discovery of tuberculin will be ranked among the most valued of the many gifts mankind has already received from Robert Koch.

Professor R. KOCH.

When in the year 1890 I made my first exact communications regarding tuberculin, I was able to point to two important properties of this medicament —namely, its power to produce specific reactions in persons suffering from tubercular disease, and its therapeutic efficacy if used for a length of time.

With reference to the former property I expressed myself in the following words: "I believe I do not go too far when I assume that the medicament will in future be an indispensable aid to diagnosis. It will enable one to diagnose doubtful cases of incipient phthisis even when one fails to obtain certain knowledge of the nature of the disease by finding bacilli or elastic fibres in the sputum or by the physical examination."

As to the therapeutic effect of tuberculin, I said on that occasion: "The main thing in the new treatment is *that it begin as early as possible.* The incipient stage of phthisis is to be its proper aim, because it is against that it can fully develop all its power." And in another place: "After this experience I am disposed to believe that *incipient* phthisis can be cured by the medicament with certainty."

Since that time I have had very frequent opportunities of testing the efficacy of tuberculin, and have invariably been able to convince myself anew of the correctness of the statements I made then. I therefore still adhere to the opinion that tuberculin is an indispensable aid to diagnosis and a very effective remedy for incipient phthisis.

In proof of the diagnostic value of tuberculin I point to its extensive use as a means of ascertaining tuberculosis in cattle. According to the calculations which Voges has collated from numerous reports, and according to the careful investigations of Eber, it gives, if properly applied, correct diagnoses in ninety-seven to ninety-eight cases in a hundred. Considering that the diagnosis is made by a single injection of tuberculin, and that errors caused by accidental rise of temperature due to other causes are not entirely excluded, this is a splendid result. Moreover the injection of tuberculin into cattle, of which we may safely say that it has now been performed in millions of cases, has shown that it is absolutely free of danger; at least, not a single case of its having caused any injury to health has ever come to my knowledge.

In the case of human beings, however, the conditions of the diagnostic use of tuberculin are considerably more favourable, for it is not necessary to extort the diagnosis by a single injection, and therefore we need not give so large a

Digitized by Google

dose or produce a strong reaction. On the contrary, we may rest content with a quite slight reaction, but must then repeat the test, in order to exclude the possibility of error. For this purpose I use the following method. In the first place the patient's temperature is observed for at least one day, or better, two, in order to ascertain whether the temperature is below 37° C. Patients whose temperature is above 37° are unsuited for the diagnostic application of tuberculin, and ought not under any circumstances to be subjected to the tuberculin test. If a patient is found suitable he receives an injection of tuberculin under the skin of the back in the forenoon; with weak patients one begins with one-tenth of a milligram; with stronger ones, whose tubercular alterations are probably very slight, one may begin with one milligram. If there is no rise of temperature at all, one gives a dose double as large as the first, not on the next day, but on the day after the next; but if there is a slight rise of temperature—only a quarter of a degree, for instance—the dose is not raised, but repeated as soon as the temperature has gone down to its normal level. It very often happens that, though the same dose has been given, the second reaction is stronger than the first. This is quite specially characteristic of the effect of tuberculin, and may be regarded as a quite infallible sign of the presence of tuberculosis. But if the first small doses produce no reaction, one gives five, and finally even ten milligrams. For certainty's sake I am accustomed to give this latter dose twice, and only when no reaction occurs even then do I feel justified in assuming that the case is not one of fresh or progressive tuberculosis demanding specific treatment.

If one adopts this method one will never expose a patient to danger nor even cause him any serious discomfort, for, as I have already said, slight rises of temperature, up to 38°, which give most patients hardly any disagreeable sensation whatever, suffice.

In course of time I have had a large number of diagnostic tuberculin injections performed in my hospital on the principles just stated. By the end of the year 1900 the number amounted to 2,890; if I add to these the other cases personally observed by me elsewhere, I get a total of considerably more than 3,000 cases. This is the material from which I derive my experience of the diagnostic value of tuberculin; and I have found that the cases in which, owing to indistinct reactions, a sure diagnosis is impossible are quite exceptional. As a rule, one succeeds either in obtaining distinct reactions or in ascertaining their absolute non-occurrence. In the former case one may, as is generally known, conclude with certainty that there is a tubercular focus, and unmistakable locally limited symptoms very often indicate its position. The tuberculin test is of special value in judging of catarrh of the apex pulmonis without tubercle bacilli in the sputum, especially when influenza is prevalent, because then cases pretty often occur which, to the eye of the clinical observer, exactly resemble incipient tuberculosis, but are not that. Among our numerous patients suffering from catarrh of the apex pulmonis there were almost 15 per cent. in whom no reaction took place; and in all the cases that could be observed long enough, the further course of the disease confirmed our opinion that they were not cases of tuberculosis.

Digitized by Google

Every case of pleurisy too, as soon as the fever is completely gone, and when there is no sputum containing tubercle-bacilli, ought to be tested with tuberculin. Of our pleurisy patients 73·2 per cent. reacted.

I wish expressly to add that I have never seen any disadvantageous effect of tuberculin when it was used in the way I have described.

The conviction that pulmonary tuberculosis in its earliest stages is curable, and that it is therefore specially necessary to recognise tuberculosis with certainty in its first beginnings, has been continually gaining ground of late, and the use of tuberculin as a means of diagnosis has again become more and more frequent. In several German sanatoria, for instance, its application has been introduced, and I believe that just these establishments, to which it must be a matter of very great importance to get their tubercular patients in as curable a condition as possible, will find the tuberculin test to be an aid of quite special value.

I come now to the therapeutic value of tuberculin, and of this too I maintain that it is completely proved, provided—and on this I have insisted from the first—that its application be restricted to still curable cases, *i.e.*, to those which are not yet too far advanced, and not complicated with other morbid processes caused by streptococci, staphylococci, pneumococci, influenza-bacteria, etc. As these processes are almost always accompanied by rises of temperature, the best way of guarding against the misapplication of tuberculin is to use it only in cases in which the temperature of the body does not exceed 37° C. That tuberculin exercises an exceedingly favourable influence on all such cases, and even completely cures them as a rule, is a fact of which I have repeatedly convinced myself, and a number of other medical men who have studied the therapeutic value of tuberculin for years, and have either published their experience of it themselves or communicated it to me privately, have arrived at the same result. As such I name Spengler, Turban, Petruschky, Krause, Thorner, Heron, Rembold, Bandelier, Goetsch, Kirchner, and Kartulis, to whose publications I beg to refer you.

With regard to the therapeutic application of tuberculin, however, it is a fact of special importance that the producing of strong reactions, such as were deemed necessary at first, is now generally abandoned. On the contrary, physicians endeavour to keep the reactions as slight as possible, and not to repeat an injection of tuberculin until the preceding reaction has completely passed off, and the temperature has been normal again for one or even several days. One can even, as Goetsch has shown, carry out the treatment without any reaction at all. It is also very expedient to repeat the treatment with tuberculin, with intervals of three to four months, till the capability of reaction is permanently extinct. Petruschky, who has given this method the name of "interval treatment," has effected by it cures whose permanence has stood the test of years of observation.

The rules which experience has prescribed for the treatment with tuberculin may therefore be briefly summarised as follows:

1. Only patients that have no fever, and in whom the process has not advanced too far, are suited for the treatment.

Digitized by Google

2. One begins with a very small dose, and increases it so slowly that only very slight reactions, or even none, take place.

3. If reactions take place, tuberculin must not be injected again till the temperature has been normal for one or several days.

4. The treatment with tuberculin must be repeated till, after an interval of three to four months, the capability of reaction is permanently extinct.

THE CHAIRMAN (SIR R. DOUGLAS POWELL).

I am sure we are greatly indebted to Professor Koch for coming here to enlighten us on this very important question of the treatment of tuberculosis and the importance of tuberculin in the matter of diagnosis and therapeutics. I think the two questions which seemed to arise very much out of the two able papers that we have just listened to are those of diagnosis and treatment. It has struck me that a matter of great importance is the statement of, I think, Dr. Heron and Professor Koch—that the tuberculin should be used for diagnosis only in cases where the temperature is normal, and should not be repeated until the temperature has again become normal. But, unfortunately, in our practice we find that the cases of tuberculosis which come before us already present an abnormal temperature, and it is under these conditions that we wish to know how to act. The same point applies to cases where the tuberculin is to be used in active disease. Again, here it is our great difficulty to get the temperature normal. As soon as it gets so we are only too pleased to let it remain so; and we want to know, in reference to the use of tuberculin, whether, when the temperature has become normal, we are still to go on treating the case with doses of tuberculin. Then the question of mixed infection comes in—mixed infection which we know to be so very important, and which is becoming increasingly recognised as of great importance in tuberculous affections. We are told that under these circumstances tuberculin is not, as I understand, applicable.

These are two or three points which strike one as those which may be very usefully dealt with in future speeches, and upon which I hope we may receive further information from Dr. Heron and Professor Koch in their answers.

PROFESSOR OSLER, *Professor of Medicine Johns Hopkins University, Baltimore*,

Regretted that he had not with him the statistics from the Johns Hopkins Hospital, in which for the past six years tuberculin has been used in the medical and surgical departments. From the latter they have been published, and show the great value of the method in tuberculosis of the bones. "In the medical wards we have used tuberculin very much as Professor Koch has advised, and I bear willing testimony to its inestimable value in certain cases, particularly in doubtful apex lesion, in obscure abdominal cases, and in pleurisy. An important point is its harmlessness. I remember no cases in which injurious results have followed the injection."

Digitized by Google

He had had no experience of late with the use of tuberculin in the treatment of the disease. Like the chairman, he had been impressed with the strong statement of Professor Koch, that only cases with a normal temperature are suitable. "There are instances of pulmonary tuberculosis afebrile from the start, and continuing so with progressive active lesions, yet in a large proportion of the cases when the fever has disappeared we congratulate ourselves that the patient has reached a favourable stage. The choice then between the use of tuberculin and the modern open-air treatment would, I think, be decided by the cleverest physician very strongly in favour of the latter."

PROFESSOR B. FRÄNKEL, *Berlin.*

I CONSIDER that I must comply with the President's request that has just reached me to speak in reference to tuberculin, because the discussion which followed my lecture, delivered to the Berlin Medical Society in 1890, ushered in the decrease in the use of tuberculin. Since that time, however, I have never ceased to employ tuberculin. I believe that the time has now arrived when the pendulum of public opinion is once more swinging to the opposite side, and it will be possible to resume the application of tuberculin without common prejudice keeping the patients in a condition of continual apprehension. I can say of some of my cases, treated and improved by tuberculin in the year 1891, that they are quite well at the present time. The avoidance of fever must be the paramount consideration in the use of tuberculin for therapeutical purposes in suitable cases. This necessitates great patience at the commencement; the small doses constitute a temptation to proceed too fast. The percentage is what the increase must depend upon; 100 per cent. are from one milligram to two milligrams, only 20 per cent. from four decigrams to five decigrams. In regard to the employment of tuberculin for the purposes of diagnosis, I have already given my views. It provides us with an earlier means of certain determination concerning tuberculosis than by any other method, and is on that account an essential condition of successful therapeutics.

DR. DENYS, *Louvain.*

CETTE communication a rapport à des travaux qui sont la continuation de ceux de M. Broden, élève de l'Institut de bactériologie de Louvain, et qui ont été publiés dans les Archives de médecine expérimentale de 1899.

Si on injecte dans le péritoine des chiens une dose convenable de bacilles de la tuberculose, on est sûr de les rendre tuberculeux et de les faire périr de tuberculose péritonéale, compliquée de tuberculose généralisée aux poumons au foie, à la rate.

A l'autopsie, on ne trouve pas de tubercles dans le péritoine ou c'est à peine si on en trouve de tout petits.

Digitized by Google

Dans ce dernier cas, il faut examiner le péritoine avec attention, tellement les néoformations tuberculeuses sont menues.

Par contre les lésions inflammatoires sont très marquées : il y a un exsudat abondant, la séreuse est épaisse, blanchâtre, et l'épiploon est transformé en une masse compacte, infiltrée de pus. Il ressemble souvent à une éponge dont les mailles seraient remplies de sérosité purulente. Les bacilles sont extrèmement nombreux.

En résumé ce qui caractérise la lésion péritonéale chez les chiens inoculés, c'est la grande quantité de bacilles, l'intensité des phénomènes banals de l'inflammation, l'absence de tubercules ou la présence de tubercules à peine ébauchés.

Si l'on inocule des chiens de la même façon, mais si au lieu de les abandonner à eux-mêmes, on les soumet à des doses croissantes de bouillon de tuberculose séparé des bacilles par filtration et non chauffé, la lésion péritonéale subit une évolution différente.

La lésion se caractérise par un développement de beaux tubercules gris, secs, qui sont surtout nombreux et volumineux dans l'épiploon. Les infiltrations purulentes ne se produisent pas. L'exsudat disparaît ou est peu abondant. Le nombre des bacilles est considérablement inférieur à celui des chiens non traités.

En résumé, chez les chiens injectés avec le bouillon de tuberculose filtré la manifestation prédominante est la formation de nombreux tubercules bien développés. Les manifestations banales de l'inflammation : exsudat abondant, infiltrations purulentes, font défaut.

Comme les chiens traités à la tuberculine présentent une évolution plus lente de la tuberculose et qu'un grand nombre d'entre eux guérit, il y a lieu de considérer la formation du tubercule comme une manifestation de résistance de l'organisme.

Dr. DENISON, *Denver, Colorado, U.S.A.*

Before reading my paper (page 117) I wish to explain where, in the ordinary treatment of tuberculosis, I would place tuberculin, and in what order of precedence I would give that or the antitoxine treatment. To make it plainer I have written on the blackboard the ratio, which is of course approximate, and would probably be changed by every physician; 100 per cent. represents the combined methods of treating tuberculosis. I myself would place 25 per cent. to 35 per cent. of what we can do for tubercular cases to the credit of climate, and 15 per cent. to exercise and outdoor life. These two would be interchangeable in the mind of the physician, and they show one-half of what we can do for our patient; 15 per cent. I would attribute to feeding and attention to the alimentary canal; 15 per cent. to individualisation—that is to say, what the physician himself can do; 15 per cent. I would give to surgical interference in healing local trouble. We have then remaining from nothing to 20 per cent. due to the process of immunisation by the antitoxine treatment. Some cases are not suitable at

Digitized by Google

all, and others can get, I believe, the full amount of good we can do for the tubercular cases. I do not propose to read the whole of my paper, because it would simply be going over the ground which Professor Koch has so excellently given you. I will summarise by saying that, considering the diagnostic effect and the second immunising effect, I have given the composition of the different preparations of tuberculin that have been made, and have also introduced the tables of our results in the United States, including about 1,200 cases from Dr. von Ruck, of Ashfield, North Carolina, and over 200 of my own. I simply wish to read the conclusions of the paper, so as not to take up your time, chiefly touching on the process or *modus operandi* of the immunising effect.

DR. C. THEODORE WILLIAMS, *London.*

THE discovery of tuberculin was unfortunately shrouded with secrecy for a time, but this secrecy was fortunately removed at a later date. With regard to its diagnostic value I think there can be no doubt, for the way in which tubercle in any part of the body, and notably in the lungs, is lit up after injection of a small dose of tuberculin, is wonderful. Around old pulmonary lesions, which had been forgotten, this insidious agent causes crepitation of a sonorous character to be heard, and lupus spots show irritable surfaces; so that it is no wonder that our imaginations were captivated by the penetrative power of the agent, and we fondly thought we had only to modify it, or incorporate some remedial agent with it, and we should reach tubercle and deal with it effectually; but when we consider it therapeutically the advantages are *nil.* The Brompton Hospital report of the patients treated according to Professor Koch's directions showed very clearly the following :—

I. That in early tuberculisation (with no mixed infection) the use of the tuberculin led to breaking down of the tubercular masses and of the lung tissue in the neighbourhood, and thus to the formation of cavities. This was proved by (1) the appearance of lung tissue in the sputum where it was previously absent, (2) by the physical signs of cavity replacing those of consolidation.

II. That in cases of excavation its use was followed by extension of excavation and spread of tuberculosis.

III. That in some cases the cavities, when formed, had a tendency to contract, and that tuberculin seemed to favour, under these circumstances, the growth of fibrosis.

IV. That tuberculin was strongly contra-indicated in pyrexial cases, and that by this means intermittent pyrexia was converted into continuous pyrexia.

V. That tuberculin did not diminish the number of tubercle bacilli in the sputum, which were often increased, and that the reaction caused by the treatment was exhausting to the patient.

Professor Koch's recommendation that it should be used in initial cases of phthisis without fever does not help us, as these are the patients who are

Digitized by Google

improved, and often undergo arrest of the disease under a variety of treatment, whether hospital, sanatorium, climatic or the like, and so tuberculin is not wanted. But does it seem reasonable, after all our efforts to build up the tissues and textures of the human body by food, tonics, hygiene and exercise, to employ means to cause a breaking down of the said tissues?

T. McCALL ANDERSON, M.D., *Regius Professor of Medicine in the University of Glasgow.*

As many other gentlemen may desire to take part in this discussion, I have put upon paper, very shortly, some of the conclusions at which I have arrived in reference to tuberculin. And in the few remarks which I have to make I shall deal with the subject under three heads:—

1st. Is it safe to use it?
2nd. What is its diagnostic, and
3rd. What is its therapeutic value?

Under the first head my experience tells me that its *use* is quite safe, while its abuse is fraught with danger, especially in the case of pulmonary tuberculosis.

For myself, I never now commence with a dose exceeding $\frac{1}{2}$ c.c. of 1 in 1000, and I am not in a hurry to increase the dose, having frequently observed that when the same dose is given twice running, the second may have a much more powerful effect than the first.

I understand it to be the opinion of some that the dose should be increased as rapidly as safety permits, so that the stage of tolerance may be reached as early as possible. But my view is exactly the reverse of thi holding, as I do, that we should defer to as late a day as possible the time when no reaction is obtained, and why? Because when there is no local reaction there is no therapeutical result. For how does tuberculin prove beneficial? When it is injected it soon ferrets out the disease, it goes straight to its goal, where it produces inflammation, and the greater the local reaction the greater the effect. On the other hand, the less the constitutional disturbance the better for the patient; but unfortunately the latter is too often proportionate to the former. So that, in our desire to obtain decided local symptoms, we must beware of producing an undesirable amount of febrile disturbance.

2nd. What is its diagnostic value?

In recent discussions which have taken place on the means to be adopted for the prevention of tuberculosis, great stress was very properly laid upon the necessity of preventing the sale of tuberculous meat, and, above all, of the milk of tuberculous cows, and with practical unanimity it was conceded that

Digitized by Google

the tuberculin test was a very certain means of ascertaining the presence or absence of tubercular disease in animals.

How is it, then, that veterinary surgeons confidently rely upon the tuberculin test for the diagnosis of tuberculosis in animals, a view which is admitted by the medical profession generally, while not one in a hundred takes advantage of it in the case of human beings when there is a doubt as to the diagnosis?

I content myself with one illustrative case out of many:—

On June 2nd, 1899, a lad, aged 11 years, came under my care in the Western Infirmary of Glasgow, complaining of weakness and of a swelling of two months' duration on the left side of the neck. His father and mother and nine of a family of fourteen were alive, but one of these was suffering from consumption of the bowels, and one from decline. The five who died succumbed to chest complications in connection with whooping-cough. Two months before admission, the patient, who had always been pale and delicate, fell off a wall, and immediately thereafter began to complain of pain in the left side of the neck, where a distinct swelling made its appearance. He was sick for a time after the fall, but in a few days the pain in the neck disappeared; the swelling, however, remained, although, according to the mother, it varied in size from day to day, and a week before he came to me it rapidly increased. The patient, on admission, was emaciated, pale, and anæmic; the hæmoglobin was 45 per cent., and the corpuscles numbered 4,000,000, there being no excess of white corpuscles. There was no fever, no œdema, and the urine was normal; but he complained of shortness of breath, palpitation, and pain across the abdomen on exertion, with occasional vertigo. The spleen was slightly enlarged, though its lower edge could not be felt, and the liver extended for an inch below the costal margin in the nipple line. There was slight enlargement of the inguinal, axillary, and submaxillary glands. The left submaxillary glands were greatly enlarged, forming a prominent mass in the left side of the neck, but the individual glands could not be distinguished. This mass was painful, freely movable, and firm. He was sent to me as a case of lymphadenoma, but I suspected that the disease might be tuberculous, and therefore tried tuberculin. The first four injections of $\frac{1}{3}$ cubic centimetre, 1 in 1000, old tuberculin, sent the temperature up to 102·2° F., 102·8° F., 102·6°, and 102·4°, while a subsequent injection of one cubic centimetre, 1 in 1000, produced a temperature of 104·8°. There being, therefore, no doubt as to the nature of the case, he was transferred to the wards of one of my surgical colleagues.

Per contra, a girl, aged fourteen, came under my care on the 14th of June last, suffering from an eruption upon the chin. This commenced about a year ago on the chin and upper lip in the form of "little white spots" which gradually spread. This was associated with copious exudation on the surface which dried up into crusts.

Under treatment the outbreak on the upper lip disappeared, leaving scars, while that upon the chin persisted, and three weeks ago the lower lip became similarly affected.

Digitized by Google

On admission the whole of the chin and lower lip were covered with very thick, adherent, brownish crusts, measuring transversely 3½ inches and vertically 2 inches, and there was a considerable amount of thick purulent exudation. The lower lip was everted, and on removing the crusts the whole surface was the seat of superficial ulceration, spreading in segments of circles and with an ash-grey base.

She was sent to me as a case of lupus, but I suspected that it might be a manifestation of hereditary syphilis, and, on further inquiry, I ascertained that about two years ago she had an ulcerated throat which continued for about twelve months; and also that shortly before this she complained for about three months of pain over the middle of the right shin, especially upon exertion or when the part was touched, and in this situation a distinct node was discovered.

With the view, however, of testing the accuracy of the previous diagnosis, four injections of tuberculin were given at intervals of a couple of days, the first two of ½ and the last two of 1 cubic centimetre of 1 in 1000, but without any trace of either local or constitutional reaction. She was therefore put upon iodide of potassium, gradually increasing up to one drachm three times a day, while the parts were dressed with yellow wash, and within a month the ulceration was gone.

It is to be hoped that the time is not far distant when, for purposes of diagnosis in doubtful cases, the injection of tuberculin will be resorted to as universally as the administration of antisyphilitic remedies when there is a question as to the existence of some obscure syphilitic affection.

3rd. What is its therapeutic value?

Here let me say at once that nowadays advanced phthisis is never subjected to the tuberculin treatment in my wards, and I fancy that, in this country at all events, there is a consensus of opinion against its employment in such cases. But in its early stages excellent results may be obtained, of which I venture to give one illustration—a case which is interesting in several other respects.

A man, aged twenty-eight years, was admitted in the second week of January, 1891. He had had five attacks of hæmoptysis within thirteen months, occurring at intervals of two or three months, and he had two anal fistulæ of nine months' duration. An examination of the chest showed entire absence of physical signs with the exception of *weakened respiratory murmur at the right base.* He was treated with the old tuberculin; thirteen injections were given in all, the first of 2 cubic centimetres of 1 in 1000 on January 16th, and the last, fifty times stronger, on February 17th. Before dismissal on March 13th the following report was made: "He has had no hæmoptysis for six months, and *râles which appeared along with much pain at the right base* after the second injection gradually disappeared within a fortnight, and there are now no physical signs. Both fistulæ have completely healed. There

Digitized by Google

has been great improvement in the general health, with an increase in weight of nine and a quarter pounds."

But it is a question, about which it would be premature at present to dogmatise, whether the tuberculin treatment is calculated to yield better results than those which have accrued from the so-called "open-air treatment," or whether the two methods might not be advantageously combined.

There can be no doubt, however, that such a combination is calculated to yield excellent results in the case of external tuberculosis, and that, in some of them at least, the open-air treatment is incapable, unaided, of removing the disease.

There can be no question of the influence of tuberculin upon external tuberculosis, and these are the best cases for studying its *modus operandi*, because *we can see the results* which follow its administration. Some little time ago one of my resident medical officers (Dr. John Aitken), at my request, kindly went over my hospital books in the Western Infirmary of Glasgow, and tabulated the particulars of forty-five consecutive cases of external tuberculosis, and without any preconceived opinions. The following is a brief summary of the results of these forty-five cases:—Thirty-five were illustrations of lupus vulgaris, seven of scrofuloderma, and three of lupus erythematodes. Of the thirty-five cases of lupus vulgaris, the duration of the disease on admission was, in one case, as low as five months; while in another it had been present for no less than forty-six years, the average duration being seven and a half years. The duration of the tuberculin treatment varied from seven weeks to six months, but one patient, who was much improved when she left the hospital, could only be induced to remain four weeks. The average duration of the treatment was four months. In thirty-three of the thirty-five cases the results were thus tabulated by Dr. Aitken: Improved, two; much improved, eight; very much improved, seven; almost well, four; well, twelve; total, thirty-three. It will thus be observed that improvement was invariable, and the number of cases dismissed well would have been much greater had we been able to induce the patients to remain longer under treatment.

Of the seven cases of scrofuloderma, the duration of the disease varied from four to thirteen years, the average being nearly nine. The duration of the tuberculin treatment varied from three to five and a half months, the average being nearly four. The results were thus tabulated by Dr. Aitken: Improved, one; almost well, one; quite well, five.

The three cases of lupus erythematodes all occurred in females. The first of those patients was thirty-one years of age on admission, the nose and left cheek being involved for a period of fourteen years; the second was eighteen years old, and the disease had implicated the face, ears, hands and arms for two years; while the third, aged twenty-three years, had suffered from it for three years, the cheeks, head, and knuckles being implicated. The duration of the tuberculin treatment was two and three-quarter months, two and a half months, and one and three-quarter months, and they were all dismissed well. In all of them the local and constitutional reactions were well marked. These results will be a surprise to many, and are even to

Digitized by Google

myself, because I have not been equally fortunate in other cases of the same disease, but one positive outweighs many negative results.

From an early period of my professional life, I have always taught that lupus vulgaris and lupus erythematodes were manifestations of the strumous diathesis, a view which was long opposed by the majority of experts. At the present time, however, the tuberculous nature of lupus vulgaris is generally conceded, while lupus erythematodes is almost universally considered to be independent of the strumous diathesis. The main grounds for this opinion are that the tubercle bacillus has not been found in the latter affection, and that tuberculin injections yield no reaction and do no good. The cases, therefore, which I have just referred to are of special interest, as supporting my view that lupus erythematodes is a member of the tuberculous family.

Now whether is tuberculin—as far as its therapeutical effect is concerned—to be regarded in the light of a constitutional or a local medication? That it produces constitutional symptoms we know, as witness the fever, the headache, and the sickness which so often follow its administration; but these are injurious rather than otherwise, and the less we have of them the better. That it produces marked local symptoms we see in cases of external tuberculosis, for we observe after each injection redness, swelling, serous exudation, and crustation, accompanied by elevation of temperature at the part; and I think it cannot be doubted that it is this inflammatory action which puts the enemy to rout, just as an attack of erysipelas of the face often improves, if it does not remove, a tuberculous affection of that part. If this be true, then it follows that we must regard tuberculin treatment in the light of a local remedy, but one which does not rectify, but rather intensifies, the constitutional vulnerability. From which it is obvious that, as we aim at permanent results, we must not rest content with injecting tuberculin only, but must combine it with general treatment.

Some of my professional brethren who have carried out the tuberculin treatment on a considerable scale—especially in the case of external tuber culosis—agree with me as to the immediately beneficial results of such a course of treatment, but state that, after the course is completed, there is generally a recrudescence of the malady. Now I freely admit that if patients are treated by means of tuberculin injections alone, and then dismissed from hospital to miserable homes, where they are deprived of light, pure air, abundant food, and tonic treatment generally, a recrudescence of the disease is all but certain. For we must never forget that two factors have to be taken into account in dealing with tuberculous disease: (1) the tubercle bacillus and its toxins, and (2) the soil favourable to its life and development. So that, in order to obtain permanent results, it is obvious that, in addition to the use of tuberculin, means must be taken simultaneously to change the soil upon which the micro-organism flourishes, such as good and abundant nourishment, living as much as possible in the open air, and the use of tonics, cod-liver oil, and other antistrumous remedies. This combined treatment, if judiciously carried out, has, in my experience, yielded excellent results, and I commend it to you with confidence.

Digitized by Google

DR. VICARY SNOW, *Bournemouth.*

MR. PRESIDENT, LADIES AND GENTLEMEN,—Of the diagnostic value of tuberculin we are all in agreement, and much that I could have said to you on this point has been already ably placed before you. Of its therapeutic value I rise with some diffidence to address you. My experience is so different from that of my friend Dr. Theodore Williams, with his larger opportunities in London. My first three cases under the old tuberculin treatment I was able to keep under observation for several years; in the first, an early shape, no symptoms or physical signs could be detected after a fever; the remaining two were doing badly before the treatment, but gradually improved both in general health and physical signs, and were able to follow their occupations, and remained well for upwards of two years. Under careful conditions I hope that tuberculin may be used in early cases as an adjunct to the very successful open-air treatment of the disease.

DR. E. C. BOUSFIELD, *London, Bacteriologist, Camberwell Borough,*

Narrated cases in which, though the "temperature canon" had been violated, the temperature of the patients gradually fell to normal during the progress of the injections, the night sweats also disappearing, and these conditions were maintained until death. Also narrated one case in which five years after the tuberculin treatment the patient was alive and well. Enquired what was to be understood by "tuberculin"; was the fluid to be obtained from a human or from a bovine source? Could the former be used for diagnosing cases of tuberculosis in the latter, and conversely? And might it not be necessary, after the events of yesterday, to distinguish the two by special and distinct names?

DR. W. M. HAMILTON, *Lancashire.*

MR. PRESIDENT, LADIES AND GENTLEMEN,—In the discussion of the value of tuberculin there is an important point to which sufficient attention has not yet been directed. I refer to the state known as a favourable or suitable soil for the bacillus tuberculosis, and would ask Dr. Koch whether he had definitely ascertained the conditions, their mode of action, and the results of that action, termed a favourable soil, when he attributed so much etiological importance to his great biological discovery. I contend that, if adequate attention had been directed to this point, we should have recognised their preponderating influence in the production of pulmonary tuberculosis, and acted on that knowledge.

If you, sir, will permit me, I should like to state in a few words the results of my investigation of the soil. At the meetings of the British Association for the Advancement of Science, and elsewhere, I have shown that a

Digitized by Google

favourable soil in man is that state of body in which there is a respiratory surface of the lungs inadequate to perform their ordinary functions, and thereby tending to accumulate in the system a substance, normally formed in the body, that possesses irritant and toxic properties; that the conditions that produce a favourable soil are those that directly and indirectly tend to reduce the respiratory surface of the lungs, and that a favourable soil can be made an antagonistic soil by subjecting the lungs for a sufficient period to conditions that directly and indirectly tend to develop their respiratory surface.

I will only add that the Polytechnic Physical Development Society has completely demonstrated the practicability of developing the lungs, and the immunity it conveyed to those members who were engaged in the phthisiogenic trades and occupations.

Dr. A. K. STONE, *of Boston, U.S.A.*,

Said in substance that he wished to make a protest against the indiscriminate use of tuberculin as a means of diagnosis, especially in cases in private practice; that the basis of his position was a personal prejudice, derived from the fact that in 1891 he himself reacted to the tuberculin test, having neither at that time nor since any symptom of tuberculosis. Becoming interested from this time on, Dr. Stone had followed the work of the veterinarians with great care, and had found that, in very many of apparently healthy cattle reacting to the test, the lesion was limited to a small, almost always entirely calcified gland. From this and the observation of many cases injected by others, and from the literature, he feels that small inconsequential lesions may give very misleading information in important cases. Careful consideration of the details of the clinical condition and of the various laboratory helps at our command make the necessary use of tuberculin very small.

Of the therapeutic value of tuberculin the observation of two series of cases treated with both the old and the new tuberculin are in the main in accord with the ideas expressed by Dr. Williams.

Dr. ERIC FRANCE

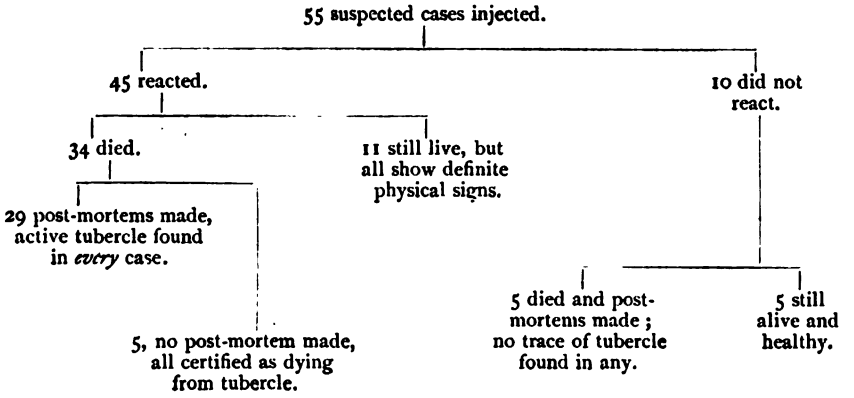
Stated that at the Northumberland County Asylum he had used tuberculin for diagnostic purposes only, with the most satisfactory results. The method he adopted was this: The suspected patient was put to bed and his temperature taken. Next day, with rigid asepsis, 1 c.c. of a ·001 solution (made by diluting 1 c.c. of Koch's original tuberculin with a ·5 per cent. solution of carbolic acid) was injected subcutaneously, and the temperature taken every three hours. If the temperature rose 2° or more within the next twelve hours tubercle was diagnosed.

Dr. Eric France injected in all seventy-five cases with tuberculin, and the results satisfied him, not only as to the accuracy of its diagnostic power,

Digitized by Google

but also as to its entire harmlessness both in the tubercular and in the non-tubercular.

Of the seventy-five cases injected, twenty were non-suspects, and were used as control experiments; in *none* of these was the faintest reaction obtained. The remaining fifty-five cases were suspected of having tubercle, and gave the following results:—


55 suspected cases injected.
45 reacted.
10 did not react.
34 died.
11 still live, but all show definite physical signs.
29 post-mortems made, active tubercle found in *every* case.
5, no post-mortem made, all certified as dying from tubercle.
5 died and post-mortems made; no trace of tubercle found in any.
5 still alive and healthy.

DR. J. E. VIVANT, *Monte Carlo.*

EN ce qui concerne la valeur thérapeutique de la tuberculine, je suis tou à fait d'accord avec M. le Dr. Th. Williams.

J'estime que ce n'est pas dans la tuberculose au début que le traitement présente le plus de difficultés.

A cette période le traitement par la cure d'air pur et de suralimentation avec tous les moyens que nous avons à notre disposition pour rendre l'assimilation aussi parfaite que possible, donne, d'après les statistiques des sociétés d'assurance allemande, 60 % de guérisons immédiates et 36 % de guérisons ayant persisté après 11 ans dans la classe ouvrière.

Ce qui nous serait le plus précieux serait un traitement spécifique qui nous permettrait de lutter à une période plus avancée, lorsque la fièvre vient diminuer rapidement les forces de l'organisme.

La tuberculine nous fournit-elle ce moyen? Tel ne paraît pas être le cas à l'heure actuelle. J'ai très peu employé la tuberculine TR, car les malades qu'on envoie dans le midi chercher le soleil et la lumière sont rarement à la période favorable d'après le Professeur Koch pour l'emploi de la tuberculine. Je n'ai eu recours à ce moyen que 3 fois.

Mais je suis resté en relation avec les trois principaux médecins d'une station qui est célèbre dans le traitement de la tuberculose par l'altitude, je veux parler de Davos Platz.

Or à Davos en 1897 le traitement par la tuberculine TR était à l'ordre du jour. Depuis cette époque le traitement par la tuberculine a baissé dans

Digitized by Google

des proportions considérables; pour représenter cette diminution par des chiffres, je dirai que cette diminution a été de plus des $\frac{1}{4}$.

Il semble donc que nos confrères ont trouvé que la tuberculine n'offrait pas à l'heure actuelle tous les caractères du traitement spécifique de la tuberculose.

As for the diagnostic value of tuberculin I should ask Professor Koch one question: Yesterday, in his most remarkable speech, he said that in his estimation human and bovine tuberculosis were quite different Is the tuberculin we have now at our disposal to be considered in the future to be effective at the same time against human and bovine tuberculosis?

Dr. EDWARD SQUIRE, *London.*

My reason for asking to address the meeting was to ask a question which, however, has already now been put by Dr. Bousfield and Dr. Vivant. "Can we use the same tuberculin for men and for animals?"

I should, however, like to make one other remark. I used tuberculin in many cases of lupus and of lung tuberculosis both in Berlin and in London in 1890, and it certainly appeared to me that the result of the injection was to cause destruction of tissue at the tuberculous focus. If this destruction of tissue, with consequent setting free of bacilli, does take place, the use of tuberculin, even for diagnostic purposes, is unjustifiable in tuberculosis of the lung.

Tuberculin is undoubtedly useful in surface or surgical tuberculosis, but those of us who have much to do with lung cases would, I think, like to feel more certain than we can do at present that its administration for diagnostic purposes is quite free from danger. If we can be convinced of its safety, its use will no doubt become much more general.

Dr. MOELLER, *Belzig, Germany.*

In my opinion it is indispensable for a medical officer of a sanatorium to employ tuberculin for the purpose of diagnosis if he wishes to take advantage of the two conditions in which its use is justifiable, that is to say, the patients must be commencing cases, and the only patients admitted to the sanatorium must be tuberculous.

The commencing cases can be diagnosed, with but few exceptions, most surely by means of experimental injections of tuberculin. I resort to injections of tuberculin in all doubtful cases in which numerous examinations by the usual methods have failed to detect tubercle bacilli in the sputum.

I can say at once that I have never observed any injurious symptoms accompany or follow tuberculin injection, with the exception naturally of such

Digitized by Google

unavoidable accompaniments as headache and general *malaise*, which, however, quickly pass away. It stands for itself that in every respect I exercise the utmost caution; the solution is freshly prepared each time it is wanted, and for injecting I employ a Pravaz syringe with a glass piston, so that it can be properly sterilised with facility.

The site that I prefer for injection is the back, where I have never noticed any local swelling follow the injection, though this is observed in most cases where the arm is chosen, notwithstanding the adoption of the same methods of sterilisation. I make the injections in the evening between 7 and 8 o'clock, so that the ensuing reaction that mostly occurs in about twelve hours can be accurately controlled on the following day by the temperature being taken every two hours. I begin with a dose of one-tenth of a milligramme. A rise in temperature of 0·5 degree constitutes, in my opinion, a reaction. An accurate record of the temperature of the preceding days is, of course, a necessity, and this is therefore furnished to me; besides, the temperature of each patient entering the sanatorium is recorded every two hours for the first eight or ten days. In those cases in which no reaction occurs with one-tenth of a milligramme, but only a rise of about 0·3 degree or 0·4 degree, then a second injection is given in about two days' time, the dose being two-tenths of a milligramme, and this in most instances is followed by a decided reaction. If after the first injection no change at all takes place in the temperature, then I give five-tenths of a milligramme in the second injection, and for the third, if there is still no rise, I give one milligramme. Intense reaction is by these precautions easily avoided; as a rule I obtain a rise in temperature of about 3·8 degrees, and I then often secure a second reaction.

I only employ tuberculin for therapeutic purposes with the full consent of the patient, and naturally only in those cases in which the general condition of the patient offers no contra-indication to its use. The increase in the dose has to be determined in each individual case, and much trouble and patience is often required to follow out the treatment. A short time ago I had a case in which fever up to 39 degrees resulted from an injection of one-tenth of a milligramme, and a further slight rise followed one-twentieth of a milligramme. Until I had for weeks injected every second day one-twentieth of a milligramme it was impossible for me to slowly increase the dose without causing fever. After about seven months I had augmented the dose to ten milligrammes, and then the case progressed comparatively quicker. I was able to give another patient the full dose of one gramme after about five months' treatment. I have been able till now to observe a favourable process of cure, *i.e.*, a continually increasing improvement in the state of the lungs, an increase in weight, with satisfactory general conditions, in all those cases in which I have employed tuberculin for therapeutical purposes. Personally, I am as yet unable to pronounce definitely on the precise degree of usefulness that should be ascribed to the therapeutical value of tuberculin applied therapeutically when combined with the hygienic aud dietetic treatment of special sanatoria. In any case, I can only reiterate that I have never yet seen any injurious effect from tuberculin. Tuberculin cannot lessen the value of the treatment

Digitized by Google

by sanatoria; on the contrary, in no case can the favourable effects of tuberculin be so well secured as by combining its application with the sanatorium treatment.

PROFESSOR SIMS WOODHEAD, *Professor of Pathology, University of Cambridge.*

THIS is a discussion which must be very largely on clinical lines, and I do not feel competent at present to give any definite opinion on this question. Therefore I shall ask to be excused, because I do not wish to waste the time of the Congress. I should have been very pleased indeed to have contributed to the discussion if I had had anything to say.

DR. OTIS, *Boston.*

I DESIRE to refer only to two aspects of this subject which I think have not been touched upon. First—It is known that syphilis will react occasionally to tuberculin, consequently in using that test for tuberculosis, one must always bear in mind this fact. From my own recent investigations regarding reactions in syphilis (published in the Transactions of the American Climatological Association for 1901), I found that a *small* per cent. of cases of undoubted syphilis gave a reaction; of course, there was the possibility that tuberculosis may also have been present. Second—The use of tuberculin as a test of cure. Dr. Trudeau, of Saranac Lake, N.Y., uses tuberculin for this purpose—if cases otherwise considered arrested or cured do not react to tuberculin, then he considers the cure assured.

DR. HUGGARD, *Davos.*

I HAD not intended to take part in this discussion, but as most of the speakers have taken one side strongly, my small experience on the opposite side may be worth mentioning. I used the first tuberculin eleven years ago in about fifty cases. While some of these cases got well that would probably not otherwise have recovered, the results in two cases impressed me so strongly that I have since then used tuberculin only at the express wish of the patient, and never willingly. In one of these cases the disease had been practically arrested, though very slight physical signs still remained. The general health was good, and the expectoration had for some time been free from tubercle bacilli. I used tuberculin, and the reactions in every respect followed a normal course. But after a little time tubercle bacilli reappeared in the expectoration, and, so far as I know, have never since then disappeared. In the other case the patient had been in good general health for years, but always had abundant tubercle bacilli in his sputum. Tuberculin caused no reaction whatever at first, and was gradually and cautiously increased, and

Digitized by Google

then an explosion occurred. The disease appeared in an active form in another place, where it had not previously been noticed. The disease then went ahead, and the patient ultimately died of generalised tuberculosis. In spite, therefore, of the good results obtained in some unpromising cases, I feel that though there may be a great future before tuberculin treatment, we do not yet know enough about it to use it clinically as a matter of routine.

PROFESSOR NOLEN, *Leiden.*

PROFESSOR KOCH told us that we may treat with tuberculin only patients when they have no temperature above 37°. Now we know that tuberculous persons commonly show a more or less marked reaction of temperature by their exercise. I therefore might ask Professor Koch what he means by patients with a temperature under 37°. Does his statement regard patients that are resting in bed, or patients, too, that are going out of doors and at work? Secondly, we know that the temperature varies at different times of the day. Does Professor Koch mean that the temperature must be at all times of the day under 37°?

Professor Koch has told us that patients that have a temperature above 37° are not suitable for the treatment with tuberculin, as these are cases of mixed infection. I cannot believe that this statement is right in reference to what I said about the reaction of tuberculous persons after taking exercise. In my opinion it is not yet proved that every feverish reaction may be considered as a certain sign of mixed infection. It seems to me that this point, however, is of a great theoretical value in judging the therapeutical value of tuberculin.

DR. HERON, in replying on the discussion, said: Mr. President, Ladies and Gentlemen,—It is a matter the mention of which should not be altogether omitted, and which I think we shall note with pleasure, that on this occasion, when discussing a subject which might have proved a hot one, we find among us, doctors though we are, a very considerable amount of agreement in opinion. It is to be remarked that every speaker here to-day has agreed that the diagnostic value of tuberculin is now an accepted fact. It is very difficult indeed, sir, to exaggerate the value of this important addition to our knowledge. It has a bearing upon the therapeutics of the subject with which we have been dealing to-day, which is most important; for there can be no doubt that when we become expert in the use of tuberculin as a diagnostic agent, we shall be able to treat cases of tuberculosis in a very much earlier stage than we can do now. Of this, I say, there can be no doubt; and, speaking from my own limited experience in this matter, I know I have with the help of tuberculin diagnosed the presence of tuberculosis in patients who have come under my observation, which in these cases showed itself by no

Digitized by Google

physical signs I was able to detect. Now, if we think what a very great advantage this is to us and to our patients, and if we remember that we are all agreed that tuberculin is a trustworthy diagnostic agent, we shall not to-day have met and discussed this subject for so long to no good purpose, but, on the contrary, to a very good and a very useful end.

It would be presumptuous in me to stand between the Congress and Professor Koch, who, I believe, will again express some opinions on the matters we have to-day discussed; but if you will bear with me for a minute or two I will try, in few words, to summarise to some extent what has been said by some previous speakers.

In the first place, a point about which we are also agreed is that tuberculin must be used in very much smaller doses than have hitherto been given. We all know that it did harm at first—and I think that was the experience without exception—because it was used in too large doses. That was a mistake which was, I think, pardonable in the circumstances. Now, of course, it would be an unpardonable mistake.

Another thing well brought out in this discussion, and it is a matter of very great importance, is that tuberculin can be given without risk to the patient's life.

Those who take the trouble to read that remarkable contribution to the literature relating to tuberculin by Dr. Goetsch, of Slawenzitz, to which I referred in opening this discussion, will be very much struck by the fact that in the treating of his cases he had great success—a success which I do not think has been equalled by anybody, at any rate, by anybody of whom I have knowledge. If you read the report of his cases you will see that a great advantage he had lay in the fact that his patients came back to him after he first treated them. He treated them first for a few weeks or months, and then, after an interval, for a few more weeks or months—with results which will strike any unprejudiced person as being proofs of the curative value of the use of tuberculin.

Gentlemen, we have heard a great deal said about high temperatures. I have always believed, and I know it is true, that it is by no means an uncommon thing to find a consumptive, whose temperature rises considerably, get rid of that high temperature by well-directed treatment. Surely it is the experience of every physician that these high temperatures are not always permanent. I do not refer to the temperatures which go on for weeks, when the patient rapidly gets worse, and the case comes to an end; I am referring to those rises of temperature which are, so far as my experience goes, essential to the existence of tuberculosis if it continues for any material length of time. These rises of temperature can surely be got rid of, and I think every one will agree—and I hope Dr. Koch will endorse what I am going to say—that, given the excess of temperature is made to disappear, the case will then be admirably suited to the use of tuberculin. But the first step is to get rid of the high temperature.

Gentlemen, a question was asked as to syphilis. I have used tuberculin in a considerable number of people, and, amongst other, in syphilitics; but

Digitized by Google

I have never seen a rise of temperature take place after the injection of tuberculin in those who were syphilitic, but not tuberculous.

That tuberculin, properly used, is a test of cure of tuberculosis, is surely a corollary to what we have heard of its value as a diagnostic agent. If it is right that we should trust to it for diagnostic purposes, it seems to me we may also trust to it as evidence of the cure of tuberculosis. The one thing necessarily follows upon the other.

There is another question which was pointedly put to me by one of the speakers, I think by Dr. Squire, as to certain symptoms being lighted up when tuberculin is given for purposes of diagnosis, or of treatment. We find, Dr. Squire said, in the lungs, sounds which were not there before the tuberculin was given; we see around a lupoid patch a halo of inflammation which was not there before, and he asks me to speak about that. My answer is, that if we follow out these cases—I am speaking out of my own experience, which I think is also supported by the experience of others—we find, when we begin the treatment, that if there were symptoms of a little infiltration at one apex, or perhaps at both apices, if we listen after we have given tuberculin we hear sounds in the lungs elsewhere than at the apices, and these sounds were inaudible before. But we shall also find that as the tuberculin treatment is proceeded with, these sounds will disappear.

I have absolutely no experience of the kind of case that Dr. Theodore Williams described, none whatever. Of course it is a question of experience. I can speak only of what I have seen. This question was studied and discussed over and over again in my hospital wards immediately after it was first mentioned by Virchow. It happened that there visited my wards at that time men from more than one part of the world. They had all sorts of beliefs about tuberculin; but never did one of them point out an instance in which there followed upon the use of tuberculin the physical signs of the condition first mentioned by Virchow, and which were just now depicted by Dr. Theodore Williams. I speak only of hospital cases, which were checked by a considerable number of medical men, and were under constant observation, and concerning them there could have been no material mistake.

Gentlemen, I will not go on any longer, as Dr. Koch, I believe, will also reply. I am sure we shall all be very pleased to hear him once more. I take this opportunity to congratulate him upon what I think is the very valuable contribution to clinical medicine which we owe to this discussion.

THE CHAIRMAN (DR. THEODORE WILLIAMS).

BEFORE calling upon Professor Koch to reply, I may say, in answer to Dr. Heron, that the cases to which I alluded were open to anybody and everybody to see. They were under colleagues at Brompton Hospital—Dr. Green, Dr. Fowler, Dr. Sydney Martin, and others, who are surely all well known as clinicians, and their observations exactly coincide with mine. I assisted in

Digitized by Google

drawing up the report, and it was submitted to the medical committee, and they agreed to it.

Before I ask Professor Koch to reply, Sir Richard Douglas Powell requested me to ask him this question: What is the source of tuberculin upon which he relies in the diagnosis of animal tuberculosis? Is it the same as that used for human cases? I believe the same question was asked by Dr. Squire. I will call upon Professor Koch to reply.

PROFESSOR R. KOCH.

GENTLEMEN,—Dr. Heron has just summed up in a most able manner what we have heard here to-day, and I agree with him in every particular.

But you desire further information from me about the tuberculin which is now used. The tuberculin now employed universally is derived from human tuberculosis.

It seems to involve a certain contradiction, if a tuberculin from human tuberculosis also causes a reaction with cattle, although bovine tuberculosis is, without any doubt, different from human tuberculosis. But this contradiction is only apparent, for this is not an isolated instance; one finds analogous reactions which affect in like manner several nearly related micro-organisms. I would remind you of the agglutinative action of the serum of typhoid fever patients, and of those who have recovered from that disease, upon the pure cultures of the typhoid bacilli—the so-called Widal's reaction: this reaction again is not absolutely specific; it has also been demonstrated that the serum of typhoid patients causes an agglutinative action, not only of the bacillus typhosus, but also upon certain bacilli coli which have been distinguished, on this account, as paratyphoid bacilli. We have to do here, not so much with reactions of very definite limitation, as with group reactions.

I have investigated different kinds of tuberculin, first the tuberculin from human tubercle bacilli, then that from the bacilli of bovine tuberculosis, and finally that from fowl tuberculosis; even this last causes reaction both in cattle and men, but much weaker.

As we see that tuberculin has qualities of this kind, they, no doubt, are connected with its chemical properties. Concerning these, however, there is so little known that it is impossible to give a full interpretation at present.

It has interested me very much to hear of the investigations of Dr. Eric France. I consider them of the first importance. It is the first time, so far as I know, that anyone has made such a trial on man supported by post-mortem evidence. It was stated that not a single deviation from the rule was found, either in those who did or in those who did not react. Such a proof is worth more than all purely theoretical explanations.

A question has been asked concerning the body temperature. It is understood that all my examples refer, in every case which exhibited a fluctuating temperature, to people who were in bed, and had been in bed for at least one day. It is often found that sick people come into a hospital with a high

Digitized by Google

temperature, and that if they remain in bed for some days this high temperature disappears. In such cases one can give tuberculin without hesitation. I certainly look upon such a person as one who must always be treated with great caution, and I should never commence with larger doses than one-tenth of a milligram of tuberculin.

Another point seems to me to be very important. The case has been often stated as if there were an antagonism between the tuberculin treatment and the open-air treatment. Having to curtail my address, I did not deal with this question, but I think it is clear that the tuberculous patient should have the advantage of any treatment and of any combination possible. I am quite convinced that in the future one must not be one-sided in the treatment of tuberculosis, and not employ merely tuberculin or merely open-air or other methods of treatment, but that everything must be gathered together which can be of special service in tuberculosis; hence I counsel as much combination of treatment as possible.

GEHEIMRATH PROFESSOR R. KOCH, *Berlin.*

MEINE HERREN,—Herr Dr. Heron hat eben in einer ausgezeichneten Weise das kurz zusammengefasst was wir hier heute gehört haben, und ich stimme mit ihm in jeder Beziehung überein.

Sie wünschen aber von mir noch Auskunft über das Tuberculin das jetzt gebraucht wird.

Dasjenige Tuberculin welches jetzt überall gebraucht wird stammt von *menchlicher* Tuberculose her. Es scheint dies einen gewissen Widerspruch zu involvieren, wenn ein Tuberculin von menschlicher Tuberculose auch Reactionen giebt beim Rinde, obwohl die Rindertuberculose ganz unzweifelhaft verschieden ist von der Menschentuberculose. Dieser Wiederspruch ist aber nur ei scheinbarer.

Denn dieses Verhalten ist nicht vereinzelt. Man findet analogische Reactionen welche sich ebenfalls auf nahe verwandte Micro-organismen beziehen. Ich erinnere Sie an die agglutinirende Wirkung des Serums von Typhus-kranken und von Personen die den Typhus überstanden haben auf die Reinkulturen der Typhus-bacillen: die sogenannte widalsche Reaction. Diese Reaction ist auch nicht absolut spezifisch. Es hat sich herausgestellt, dass das Serum von Typhus-kranken nicht allein auf Typhus-bacillen eine agglutinirende Wirkung ausübt, sondern auch auf gewisse Colibacillen die man deswegen als Paratyphöse bezeichnet hat. Es handelt sich also hier nicht um absolut feststehende Reactionen, es sind vielmehr Gruppen-reactionen.

Ich habe verschiedene Arten von Tuberculin versucht. Zuerst das Tuberculin von menschlichen Tuberkelbacillen, dann dasjenige von Tuberkelbacillen der Rindertuberculose, auch von Hühner-tuberculose. Selbst dieses letztere giebt noch Reactionen sowohl beim Rinde wie beim Menschen, aber doch viel schwächer.

Wenn wir sehen dass das Tuberculin derartige Eigenschaften hat, so handelt

Digitized by Google

es sich dabei unzweifelhaft um chemische Verhältnisse. Über letztere wissen wir aber jetzt noch zu wenig um schon eine ausreichende Erklärung dafür geben zu können.

Es hat mich ausserordentlich interessiert von den Untersuchungen des Herrn Dr. Eric France zu hören. Ich halte sie für ausserordentlich wichtig. Es ist meines Wissens das erste Mal, dass man einen solchen Versuch am Menschen gemacht und zugleich den postmortem Befund festgestellt hat. Sie haben gehört, dass nicht eine einzige Abweichung von der Regel gefunden ist weder bei denjenigen die reagiert haben noch bei denjenigen die nicht reagiert haben. Ein solches Experiment ist viel mehr wert wie alle rein theoretischen Auseinandersetzungen.

Das ist was ich über diesen Punkt zu sagen habe.

Dann ist noch eine andere Frage an mich gerichtet in Bezug auf die Höhe der Körper-temperatur. Selbstverständlich beziehen sich meine Angaben in allen Fällen wo die Temperatur schwankend ist, nur auf solche Menschen die sich im Bette befinden und zwar mindestens einen Tag.

Man findet sehr häufig dass die Kranken mit höheren Temperaturen in das Hospital hineinkommen, und dass wenn sie sich einige Tage ins Bett befinden diese höhere Temperatur verschwindet. In solchen Fällen kann man unbedenklich Tuberculin geben. Ich betrachte allerdings einen solchen Menschen immer als einen welchen man sehr vorsichtig zu behandeln hat, und ich würde bei ihm nie mit höheren Dosen als $\frac{1}{10}$ milligramme Tuberculin beginnen.

Dann möchte ich noch auf einen Punkt zu sprechen kommen welcher mir sehr wichtig zu sein scheint. Es wird jetzt vielfach die Sache so dargestellt als ob ein gewisser Gegensatz bestände zwischen der Tuberculinbehandlung und der Freiluftbehandlung. Ich habe mich in meinen Vortrag, weil ich ihn kurz fassen musste, nicht auf diese Frage eingelassen, aber ich halte es für selbstverständlich dass wir alle die Vortheile die irgend eine Behandlung fur die Tuberculösen bietet auch anwenden und sie also möglichst combiniren. Ich bin ganz dafür dass man in Zukunft die Behandlung der Tuberculose nicht etwa einseitig gestaltet und nicht etwa blos Tuberculin anwendet oder blos Freiluft oder irgend eine andere Behandlungsmethode, sondern dass man alles zusammenfasst, was den Tuberculösen überhaupt nützen kann; ich empfehle also eine möglichst combinierende Behandlung.

Digitized by Google

THE USES OF TUBERCULIN.

By CHARLES DENISON, A.M., M.D., *Denver, Colorado.*

When Koch had announced the discovery of tuberculin, and the world was agog with expectations for the new cure of tuberculosis, this sign was said to have been posted upon the shop of an ambitious barber in Switzerland—"Corns treated and tuberculin given here." What was the conception of that barber, as to the intricate nature of the remedy and the delicate shades of disease which were suited or unsuited to its use, it would be difficult to state. The case of some physicians who, under the assumed authority of professional proficiency, deal in borrowed criticisms of everything that pertains to the subject of this paper, is a little less inexplicable. A criticism, to be of weight, should be based upon a reasonably complete personal experience.

If I can succeed in clearing away any of the clouds which befog this subject, my object in presenting the following remarks upon my own experience will have been accomplished.

We will divide *the uses of tuberculin* into two parts, (1) Diagnostic and (2) Immunising.

The diagnostic powers of tuberculin are now generally recognised and much better understood than the immunising. However, we shall learn that they are not so very different from each other as the separate purposes of their employment would seem to signify. The diagnostic effect seems to depend more upon what comes out of the bacilli—*i.e.*, the quality that resides in the culture fluid in which they have grown, while the immunising property seems to centre in the bodies of the germs, as Koch determined with his new tuberculin (R).

This diagnostic effect has thus far constituted the chief use of tuberculin—*i.e.*, the glycerine extract, originally termed "lymph." The method of manufacture is to concentrate the glycerine culture fluid at ordinary boiling temperature down to one-tenth its original bulk, after which it is filtered through porcelain. It thus contains various substances—*viz.*, a combination of toxins and proteids capable of producing intense reaction in living tubercular tissue. It includes 50 per cent. glycerine—beef extract—beef peptones and salts, and small quantities of the specific tubercle and bacilli proteids, which are liberated during the growth of the culture and during the process of the boiling and concentration of the fluid.

As to the chemical constituents of this reactionary material we will let very good authority explain. In their article on "The Bacterial Toxins" Drs. Vaughan and Cooley say—"In fact, it may be safely said that in no

Digitized by Google

case has the chemical nature of the essential toxin of any of the pathogenic bacteria been shown."

They instance Buchner's investigations on pus-producing substances in bacterial cells, stating his conclusions "that there is nothing in the whole category of decomposition products, caused by bacteria, to account for the phenomena of general infectious diseases, but that the cause of these must be looked for in the albuminates of the bacterial cell itself, and that in some cases, at least, these poisons are only set free on the death and dissolution of the bacterial cell. By this theory he explains the fact that suppuration may be caused by dead cultures of germs which when living have not the pyogenic property." *

As to the *modus operandi* of this reactionary effect we are not quite so much in the dark. It seems that there is a specific stimulation of the defensive cells of the body with reference especially to infected areas, and in proportion to the sensitiveness of the nervous system, the blood supply and advanced degree of said infection. The circulation of the blood in the lungs being proportionally more active for a given area (than for any as large portion of the periphery of the body), the most sensitive and active effects may be looked for in pulmonary tissue. The increase of the number and flocking to the affected area of leucocytes and phagocytes constitutes the specific effect which has not yet been similarly shown to occur with the use of any other substances, such as cinnamic acid, oil of cloves or peppermint, neuclin, etc., which are capable of increasing *general* leucocytosis.

This effect may be so great, with too far advanced infection, as to produce a choking off of nearly destroyed tissue; a necrotic process is thus carried to an extreme, and grave problems of elimination are forced upon the animal economy as a result. It is not, however, these extreme effects with which we intend to deal; they are always and especially to be avoided.

Hueppe, in his "Principles of Bacteriology," page 363, says—"When a local disturbance occurs as a consequence of parasitic invasion, then a reactive inflammation with the formation of a limiting wall of leucocytes takes place, an event that must be considered as a natural healing process and that often ends in recovery. Ribbert asserts that this surrounding mantle of white blood corpuscles prevents nutrient material and oxygen from reaching the bacteria, but perhaps also the white blood corpuscles disintegrate, and so become chemically active in the way pointed out by Hankin."

All of which serves to explain how intimately associated are the diagnostic and the immunity effects of tuberculin. In fact, the latter process—the therapeutic effect—is diagnostic all the way through, so relative is it to the degree and to the results of infection, and so dependent is it upon the resistance and environment of the human body.

Diagnostic proficiency, then, of a discriminating and far-reaching kind is

---

* "The Bacterial Toxins." By Drs. Victor C. Vaughan and Thomas B. Cooley. *Journal American Medical Association*, February 23, 1901.

Digitized by Google

the key to success in handling this remedy. Observance of technique is also essential.

The size of the dose hypodermically given for diagnostic purposes varies with different experimenters, who give different reasons for the course they pursue. Some prefer to give very small initial doses with intervals of two or three days between. The liability is that they will create a tolerance, and so miss the mark, the temperature rise or reaction they are after. Others prefer to give a single large dose, similar to the plan pursued by veterinarians with cattle. The danger here is the possibility of getting too severe a reaction for the case in hand. The suspicions or expectations of physicians, as regards the possible existence of infection varies so greatly that some would use this test when as little or less than one milligram would cause a reaction in an adult. In such cases I think we ought to be able to determine the diagnosis without the test. So the diagnostic dose has varied, I should say, from 1 to 30 milligrams (or of a 1 per cent. solution approximately 2 to 45 minims) of the crude tuberculin (Koch). I prefer myself to follow a middle course between the above extremes, and start with what I expect may be an effective dose and daily increase it. Let it be cumulative if it must be; we thus approach the reactionary point and learn much of the *degree* of infection. Each morning give a dose approximately double that given the previous day till you reach 20 or 30 milligrams, if you do not get conclusive evidence of infection before that time. The size of the initial dose must depend upon the physician's suspicions of infection, the age, sex and susceptibility of the subject. The individual sensitiveness of certain patients to this reaction is an important phase of this matter. I have obtained reaction to 1 milligram in a male adult, and had to reach 20 milligrams before getting the same evidence in a female, with both of whom the after course and treatment apparently proved the correctness of the diagnosis.

Therefore, the initial dose for a child or susceptible young woman I would place at from 1 milligram glycerine extract of tuberculin (standardised as obtained in America through Victor Koechl Co., N.Y., or the Bacterio-Therapeutic Laboratory at Ashville, N.C.) up to 5 or 6 milligrams (or 9 or 10 minims for an adult male), and each day nearly double the previous day's dose as before described. If the patient has kept a temperature record (taken at, say, 9 A.M., 1, 4, and 8 P.M.) for two or three days previously, any habitual rise can be accounted for. I prefer a morning hour for the test-injections, because if an evening hour is chosen a sudden rise of temperature may be missed during sleep. This rise may come in four hours if the tubercular lesion is centrally located in the lungs, or at a much later time if located in other organs or in the joints. If it happens to be incapsulated in far-off glandular structure the response may be slower still, very likely manifesting itself the following day. Any suspicious but not definitely conclusive reaction may indicate the wisdom of waiting over a day or two, and then a larger dose will determine the genuineness of the effect.

We are by no means limited to the temperature curve in determining that a reaction has taken place. A temperature rise does not uniformly occur

Digitized by Google

and it may be subordinate to the *systemic* effect. The latter is much like the sensation of the oncoming of an attack of *la grippe*; in fact, it is sometimes difficult to eliminate *la grippe* if that disease happens at the time to be prevalent. Besides the systemic reaction there is the local result in even slightly affected lung tissue, perhaps previously undetected, and one which occurs earlier there than in an advanced stage of localised disease in the same lung. This is the most helpful diagnostic indication to be obtained from the test; but one must have the discriminating ear and stethoscope to appreciate it. This, I think, is a result of the *specific action* of the tuberculin, stimulating as it does leucocytosis at the point of injury. The effort at repair, being accompanied by a certain amount of infiltration and congestion of the slightly affected tissues, produces a higher-pitched breath-sound than normal, more puerile and broncho-vesicular and quite characteristic. This local reaction is also a possible guide to the direct method of immunisation which will be considered later on.

We have not time here to discuss at length the claim that other diseases—as syphilis and leprosy—will likewise respond to this tuberculin test. The uncertainty of the relation of these diseases to tuberculosis, and the very general existence of this tuberculous dyscrasia, or latent tuberculosis, in the human family, shrouds the whole question in doubt, warranting a verdict for the test—that is, where the test works affirmatively it is nearly always safe to assume that at least latent tuberculosis exists.

These uncertainties (the difference in dose and technique employed by different observers) may account for much of the discrepancy in positive results obtained, which amount to from 60 to 100 per cent., positive or affirmative diagnosis.* My own cases, including quite suspicious cases, but only such as it was deemed could not otherwise be proved to be tubercular, have given about 90 per cent. positive results; I think only five out of forty-six were shown to be negative.

The test is of interest and very helpful to us in forestalling active tuberculosis at the very time when a cure is possible. It, as Petruschky asserts, has "the power of disclosing the presence of unsuspected foci of tuberculosis," which makes it a peculiarly valuable diagnostic resource. It is safe, and no harm need come from its cautious employment.†

The subject of the immunity-producing quality in tuberculin is next in order. The need of discovering this protective principle and separating it from the crude tuberculin has been appreciated, but the problem has not been an easy one. It remains to be learned if all has been done that is possible.

* Dr. J. M. Anders—*Journal American Medical Association*, January 12, 1901—in an article on "The Diagnosis and Treatment of the Prebacillary Stage of Pulmonary Tuberculosis," summarises 3,683 cases in which this tuberculin test has been made by different physicians, with an average of 78 per cent. positive reactions.

† Petruschky—*Verträge zur Tuberculosis-Bekampfung*, Leipzig, 1900—cites the discovery by Schreiber, "That new-born children during the first week of life did not once react to this diagnostic test, to which test a great number were subjected." This should not only strengthen our confidence in the reliability of this diagnostic method, but also weaken the claims of those who believe in the possible congenital transmission of the disease.

Digitized by Google

Koch thought he had reached the goal in his new tuberculin (R). But the *crudeness* of this necessarily doubtfully perfect *emulsion*—though we admit it to be a simple substance made from the bodies of the bacilli—did not favourably impress cautious experimenters. However, the healing or immunising property seemed to have been determined, as above expressed by Buchner, "in the albuminates of the bacterial cell itself." We will return to the consideration of this remedial agent after describing the various preparations.*

I am aware that the possibility of transmitted tuberculin being proved to be the immunising principle in these animal sera may give colour to the claim that they and the direct tuberculins should all be considered under one head. Yet the *uncertainty* of these sera, from their lack of standardisation and from the variety of elements which have to be included in their manufacture, is objectionable. The amount and virility of the toxin administered to the animal, and the time and resistance required to change it to a possible antitoxin † (as it is supposed to be done in making diphtheria antitoxin), are questionable elements which require too much abject faith in the commercial side of this matter to gain full confidence. In view of the questionable composition of an antitubercle serum, it might be well to heed Ferdinand Hueppe's warning when he says of the antidiphtheritic serum—"There is serious disappointment in store for us if we do not succeed in ridding ourselves at once of the fallacy regarding the absolute harmlessness of the antitoxins." Until we know exactly what we have in these sera, let us not confound the serum with the tuberculin treatment; we can at least assume the two to be different and not interchangeable.

After the crude tuberculin, or glycerine extract, we have Koch's *Tuberculin* (R) previously mentioned. Then we have Klebs' Tuberculocidin, one of the first efforts to improve on the crude tuberculin,‡ but of questionable utility, because probably too much of the toxin is eliminated.

Antiphthisin (Klebs') is very similar, except instead of being produced from tuberculin it is obtained from the *culture fluid* upon which the tubercle

* Lack of time, and the desire not to confound the *indirect* with the *direct* tuberculin method, must be my excuse for not here fully considering the former—the anti-tubercular sera.

† The antitoxin would seem to be created *in* the blood or body of the treated animal, for Hueppe attributes to Roux the determination of this fact. "If we administer a given quantity of toxin in the form of many small doses one after another, and in another instance inject the same quantity all at once in one large dose, the amount of antitoxin found in the animal after the same lapse of time is considerably larger in the first case than in the second. . . . The toxins, therefore, act as stimuli upon the body cells, and these forthwith contribute to the blood new defensive substances."

‡ It is made from tuberculin by precipitating with an alkaloidal reagent (usually the sodic-iodide of bismuth) throwing down many of the albuminoids which are contained in the culture fluid: the precipitate is filtered out and then the filtrate is further treated with an alkali, for the purpose of precipitating the reagent used in the first place, and is again filtered. The clear fluid is then treated with absolute alcohol, obtaining another precipitate which is collected upon a filter, dried, and redissolved in distilled water. Standarised to one per cent. the liquid contains proteids from the beef extract and peptones; it is, however, free from glycerine and from salts; contains also some of the specific products from the germ the same as does tuberculin, and as much, if none has been lost in the process of purification.

Digitized by Google

bacilli have been grown, without the application of boiling heat for concentration. This was thought to preserve in the material an immunising property in the proteid, which power would have been destroyed by boiling; otherwise this preparation is treated and standardised the same as tuberculodin.

Tuberculinum purificatum (von Ruck), unlike the two previous preparations, is made from the whole culture fluid with the tubercle bacilli grown therein included. The object being to extract more of the tubercle bacilli proteids, it is boiled at a temperature of 120° to 130° F. *in vacuo* for a long time, say for six weeks or two months. The tubercle bacilli are then filtered out and the filtrate is treated and standardised the same as in the preparation of tuberculocidin and antiphthisin. This preparation then differs from the other two in that it contains more of the specific tubercle bacilli toxins. It is not so much of a departure from the crude extract as they are, and it undoubtedly contains considerable of the principle Koch found to be immunising in his tuberculin (R). This purified tuberculin, besides being an improvement on the crude article, has this advantage over the next greater improvement made by von Ruck in his *watery extract*—namely, that it costs about one-tenth as much, and therefore should be suitable for use in practice among the poor. Koch's tuberculin (R.) undoubtedly contained the choice toxin immunising property. Six years ago a substitute in the form of a liquid extract was suggested by me to Dr. von Ruck as an equivalent which should be thoroughly filtered and standardised. The object was to get rid of the unpurified emulsion quality in the tuberculin (R.). Dr. von Ruck has well accomplished this in his watery extract, and gives his technique of manufacture as follows: No culture fluid at all is used, only the tubercle bacilli obtained from full virulent cultures. These are filtered out of the culture fluid, and while on the filter are washed with distilled water to thoroughly remove the same. Then all moisture is removed by drying *in vacuo*, when they are powdered as finely as possible in an agate mortar. After this they are treated with sulphuric ether for the extraction of fats, and this treatment is continued till fats are no longer obtained. Thereafter the tubercle bacilli are again dried and powdered as before, when they are placed in a large glass container and pure, sterilised, distilled water is added. This glass container is then placed in a continued water bath with a temperature between 120° and 130° F. for a period of two or three months, and occasionally agitated, when the fluid is syphoned off and filtered through porcelain. This fluid is then examined for proteids (and if necessary is concentrated) and standardised to one per cent., which represents the "number 100" solution. The result is that this fluid contains only the pure tubercle proteids (soluble in water) and nothing else. The whole process, including the germ culture and the after-testing on guinea-pigs, takes about a year for its completion. Experimental work has shown that the final product is the undoubted toxin of the tubercle bacillus, and that it contains immunising and curative properties.

Thus it is apparent that the study of the tuberculins, during the ten or more years since Koch put out his first product, has been fruitful in the evolution of a remedy which approaches perfection.

Digitized by Google

Table of Results of Specific Treatment.

Roman numerals—Stage of disease. *Av. yrs.*—Years known to be alive after commencement of treatment.

Specific remedy used.	Now or recently known to be in good health.	Now supposed to be living.	Whereabouts unknown: possibly dead.	Dead: cause in part known.	General Results. Disease thoroughly arrested or seemingly cured.	Much benefited, immunity increased, life prolonged.	Retrograded or died: possibly of intercurrent causes.
Tuberculin (Koch) 57 cases reported.	I. 7 II. 3 III. 7 Av. 8 1-2 yrs.	I. 5 II. 4 III. 9 Av. 2 1-2 yrs.	I. 0 II. 1 III. 7 Av. 2 yrs.	I. 0 II. 4 III. 10 Av. 2 1-2 yrs.	18	23	16
Tuberculocidin (Klebs) 21 cases reported.	I. 3 II. 2 III. 1 Av. 7 yrs.	I. 1 II. 2 III. 3 Av. 2 3-4 yrs.	I. 0 II. 0 III. 5 Av. 1 2-3 yrs.	I. 0 II. 0 III. 4 Av. 1 yr.	6	10	5
Antiphthisin (Klebs) 47 cases reported.	I. 6 II. 4 III. 6 Av. 5 yrs.	I. 3 II. 3 III. 11 Av. 1 3-4 yrs.	I. 0 II. 1 III. 7 Av. 1 1-2 yrs.	I. 0 II. 1 III. 5 Av. 2 1-2 yrs.	17	19	11
Asses' Serum (H. K. Mumford) 3 cases reported.	I. 0 II. 0 III. 1 Av. 3 yrs.	I. 0 II. 0 III. 1 Av. 1 yr.	I. 0 II. 0 III. 0	I. 0 II. 0 III. 1 Av. 2 yrs.	0	2	1
Antiphthisic Serum, T.R. (Fisch) 11 cases reported.	I. 1 II. 2 III. 1 Av. 3 1-2 yrs.	I. 0 II. 1 III. 1 Av. 2 yrs.	I. 0 II. 2 III. 1 Av. 1 1-3 yrs.	I. 0 II. 1 III. 1 Av. 1 yr.	3	5	3
Oxy-tuberculin (Hirschfelder) 3 cases reported.	I. 0 II. 0 III. 0	I. 0 II. 1 III. 0 Av. 2 yrs.	I. 0 II. 0 III. 1 Av. 1 yr.	I. 0 II. 0 III. 1 Av. 1-2 yrs.	0	1	2
Tuberculin purif. (von Ruck) 26 cases reported.	I. 5 II. 1 III. 2 Av. 3 3-4 yrs.	I. 3 II. 2 III. 4 Av. 1 1-2 yrs.	I. 0 II. 0 III. 1 Av. 1 yr.	I. 0 II. 0 III. 8 Av. 1 yr.	9	9	8
Watery Extract (von Ruck) 45 cases reported.	I. 13 II. 6 III. 9 Av. 1 2-3 yrs.	I. 2 II. 2 III. 5 Av. 1 1-4 yrs.	I. 0 II. 1 III. 2* Av. 1 yr.	I. 1 II. 0* III. 4 Av. 3-4 yrs.	18	22	5†

* None of these were completed cases, 3 died of pneumonia, 1 of *grippe*, 1 from result of dissipation, and all some months after treatment.

† Previous column should rightly have benefit of 3 of these.

Digitized by Google

Reduced to percentages, and lumping similar methods for comparison, we have the following table:—

TABLE OF COMPARISONS BY PERCENTAGES.

	Disease arrested, or apparent cures.	Much improved. Immunity relatively increased.	Retrograded: or died from various causes.	
Tuberculin	32	40	28	Approximately 7 years since treatment.
Tubercu'ocidin Antiphthisin Tuberculinum Purificatum	33	40	27	Approximately 4 years since treatment.
Asses' Serum Antiphthisic Serum	30	40	30	Approximately 3 years since treatment.
Watery Extract	40	49	11	Approximately 1 year since treatment.

My own experience favours this conclusion, and as there has been no time that I have not been at work with some one or two of these preparations, I can conscientiously say that the last is preferable to all others.

The accompanying table, completed Feb. 20, 1901, gives the general results in 213 undoubted tubercular cases, being probably one-fourth of those seen by me, and all of those I have treated with any form of tuberculin in the ten years named, excluding those not under that treatment longer than a month, also excluding those treated for diagnostic purposes only. It may be said that the gravity of the disease in these cases was sufficient to seem to demand that some additional help for them should be found beyond the usual methods of treatment. I mean even beyond the combination treatment which I always deem essential, embracing climate, hygiene, physical exercise, outdoor life, attention to alimentation, appropriate inhaling method, etc. The complications and conditions indicated gravity, which was shown by the stage of disease at commencement of treatment. Of the 213 cases reported in the table, 50 were in the first, 45 in the second, and 118 in the third stage. Note, too, that the results are carried forward to as late a date as possible, so that the favourable outcome bears evidence of the staying qualities of the immunity produced, even admitting that it was partial or incomplete. To summarise:—Of 57 patients treated with crude tuberculin 8 to 10 years ago, 17, or 30 per cent., are now known to be living; of 94 patients treated with the purified forms of tuberculin (tuberculocidin, antiphthisin and tuberculinum purificatum) from 4 to 7 years ago, 30, or 32 per cent., are living in good condition; while of 45 watery extract cases, 28, or 62 per cent., are living in apparent immunity.

Digitized by Google

For comparison I also introduce herein Dr. von Ruck's lately tabulated

COMPARATIVE TABLE OF RESULTS.*

	No. of cases reported.	Percentage.	
		Recovered.	Improved.
Treated without specific remedies	816	12·1	31·0
Treated with Koch's Tuberculin	379	35·5	37·5
Treated with Antiphthisin and Tuberculocidin .	182	32·5	56·8
Treated with purified Tuberculin (von Ruck) .	166	43·4	39·2
Treated with Watery Extract of Tubercle Bacilli (von Ruck)	303	56·1	33·7

I leave these tabulations to speak for themselves, for I wish to devote any remaining time allotted me to the discussion of the *modus operandi* of this immunising process.

Ferdinand Hueppe † says, "By undergoing a disease the predisposition to that disease is removed, and converted into its opposite immunity. In no case can anything appear in the form of disease which was not previously present in the body as a predisposition; external forces are able merely to make this predisposition apparent."

Applying this fundamental statement to tuberculosis, we may be able to successfully answer the prevalent objection to this immunising use of tuberculin. We can thus show that it is pre-eminently a natural process, and safe when intelligently used.

The objection referred to is based upon *fear*, which, in turn, is grounded upon a false conception of the nature of this dyscrasic disease. The *fear* is that by the hypodermic injections we are introducing a toxin into a system already burdened by an excess of the same poison. This is a misconception of the facts. The *truth* is, the toxin which is *in* the system is not appreciated by it in the way of being absorbed into the circulation. The whole nature of the process is to oppose such a result. Absorption is impossible, because the toxin is stowed away in the germs in non-vascular tissue. Tuberculosis is not the destructive disease we picture it, at least not until it has reached and passed the breaking-down stage. Till then it is a building-up, defensive process—*i.e.*, during the pretubercular and infiltration stages.

When this dyscrasia, which is an acquired fault of our civilisation (more fully considered in my Paper on "Devitalised-air Toxemia—a Prime Cause of Tuberculosis"), becomes a germ disease, whether by a possible auto-genesis or by transplantation from without the affected body, then the toxins are formed and centred in the bodies of the germs. If, in the beginning of their

* From "Clinical Reports," etc. Winyah Sanatorium, Ashville, N.C.

† Hueppe, *Op. cit.*, page 249.

Digitized by Google

formation, these toxins could be appreciated by the system, the disease would be auto-curative, if I may coin the term, and the tolerance or purification of the system, which we know occurs slowly under the present *régime*, would happen quickly; or if rapidly an unsurmountable amount of toxin were generated the end would also come quickly, as it does in diphtheria. But it is not that way in tuberculosis: the leucocytes and phagocytes get in their defensive warfare and stop absorption in the beginning of the trouble. They build their walls of non-vascular tissue around the germs—which encapsulation constitutes what we know as "tubercles"—and absorption thereafter is almost impossible. Not only this much hindrance to absorption is the rule, but the bacilli themselves are enshrouded in a coating of fat, which still further prevents their disintegration even should they be set loose in the blood current. It is only by intricate chemical procedure that these enveloping fats, or fats and cellulose, can be dissolved away from the germs.

Not only is there this much of natural defence against the appreciations of the toxins by absorption in the commencement of tuberculosis, but all the way through the same relief from absorption is characteristic of Nature's provision. When it comes to the breaking up, and necrotic processes and ulceration show that the non-vascular tissue has reached its limit and its death has commenced, then the vomicæ in the lungs and profuse expectoration of bacilli and mixed infection can still be looked upon as part of Nature's effort at self-defence. Degeneration and breaking down occur, but there is not enough absorption of the toxin to stimulate Nature's antitoxic power; and finally there is not enough antitoxic power remaining in the exhausted system, not enough leucocytes and phagocytes left *to be stimulated*.

What is needed is to aid Nature's defence when the cells are there to fight, and when there is not too much for those that are there to do.

There are many ways of stimulating this defence; and climate, exercise, and good feeding can do wonders, all of which nullify the injury of rebreathed or devitalised air. But for specific stimulation, when Nature has no proper direction given to her defensive cells, choose the toxin from the nucleus of the germ isolated from extraneous fats, and not heated so much in the making as to lessen or destroy its immunising power. Petruschky is satisfied with Koch's new tuberculin (R.), but he appears not to have known of von Ruck's watery extract, a more refined and better standardised product. Choose only such cases for the treatment as will favourably stand the specific stimulation. Be constantly hedging, by *under* rather than by *full* dosage, against over-action. The direction laid down by Petruschky is not bad. He says, "We should aim to produce a strong local but weak general reaction." But do not expect any immediate healing effect upon necrotic tubercular tissue, concealed tubercular abscesses, ulcerating cavities, or any other manifestation of tuberculosis where the circulation of the blood does not reach. Such conditions are obstacles to any treatment whatever, and will be favourably influenced very slowly if at all. This may mean a large concession to our average inability to accurately state what is taking place in concealed areas. Especially does extra caution need to be taken when tuberculosis is complicated with active

Digitized by Google

pleurisy, with *la grippe* or other toxæmia, as measles, etc. Better wait for the subsidence of any such conditions before proceeding with the specific treatment.

Dividing, as does Hueppe, the kinds of immunity into (1) natural, (2) artificial, and (3) toleration, it is the third we are seeking to strengthen by our specific medication. By the other two, parasites are prevented from entering the body, but by this third kind the body has acquired the power of "paralysing" the toxins of the germs. One of the best proofs possible that very little of the toxins are absorbed during the course of the disease tuberculosis is that this toleration immunity acts as it does. There is no time in this disease, from the earliest period of tubercle formation till years of the process have passed, when patients are not most sensitive to tubercle bacilli toxins. Yet experience shows that that sensitiveness can be gradually overcome, and tremendous doses of toxins finally given without any reaction whatever. This would not be so if the germs were giving up these same peculiar toxins through absorption. The acquirement of this protective immunity takes time, for it must come about through slow and laborious intervention of the body cells. As Hueppe says, "The body cells, because of their relatively great independence of varying conditions of nutrition, are able to cling to their inherited or acquired characteristics." The resistance to their action is of course relative to the extent and results of the existing toxæmia. In incipient cases of tuberculosis toleration immunity, to the extent of a cure, may be obtained by this specific method in two or three months; while in others, past the beginning of the ulcerating stage, the fight may have to be prolonged part of the time for two or three years, and that too with all the possible outside helps we can muster to our aid. Hueppe's reasoning seems to show that it is the active protective immunity, as by the direct method, which we need rather than the transient passive kind, as by the indirect method. He says, "It is the organism of the host, the cellular tissues of the human being, that reacts toward the stimulus of those bacteria or bacterial proteids or toxins with which we endeavour to heighten the resistance toward parasities and parasitic poisons. The cells eliminate this strange proteid as soon as posssible. It is for this reason that passive immunity is not lasting." The chronic nature of tuberculosis, with its incapsulation of toxic products, and its protective walls of hypertrophied and infiltrated tissues, indicates the lesser application of the transient *passive* (indirect sera) as compared with the staying *active* (direct) method of immunization. Hueppe says (p. 395), "In general, however, the defensive reaction which is provoked by isopathic or specific stimuli is the stronger and more persistent, and hence often appears peculiar in kind." It is *peculiar* in kind, and is found, as Koch, Buchner, Vaughan, von Ruck and others have surmised or shown, in the proteids from the bodies of the germs themselves.* Gamelia, Hueppe, and afterwards H. Buchner, discovered that specific poisonous substances found *outside* the parasites in the culture fluids were

* Hueppe: *Op. cit.*, page 308.

Digitized by Google

not identical with the protective substance occurring *in* the disease germs or their metabolic products. Petruschky, then, was wrong in his statement "that nothing will be gained by the search for new forms of tuberculin." So have men high in authority said that there was no use looking for any specific whatever in tuberculosis, for they said there was none to be found. Yet I, for one, like to think that there is more in store for us to be discovered, in reference to tuberculosis, than all we now know.

SUR LE SÉRO-DIAGNOSTIC DE LA TUBERCULOSE. TECHNIQUE ET RÉSULTATS.

By MM. les Drs. S. ARLOING et PAUL COURMONT.

Le séro-diagnostic de la tuberculose est actuellement trop répandu pour que nous voulions exposer ici toutes les faces de la question ; notre mémoire risquerait d'être soit trop long, soit très incomplet. Nous nous étendrons surtout sur la partie technique de la question, sur les difficultés et causes d'erreurs de la méthode et sur les moyens pratiques de surmonter les unes et d'éviter les autres.

En effet, nous avons eu le plaisir de voir confirmer la découverte de l'un de nous, et nos travaux communs, par le plus grand nombre des savants français ou étrangers qui se sont occupés de la séro-réaction tuberculeuse ; mais nous nous sommes aperçus d'autre part que quelques uns s'étaient heurtés, dans l'application, à des difficultés de technique ou à des divergences d'appréciation qui pouvaient expliquer certaines divergences de résultats ou de conclusion. C'est pour cela que nous développerons avec détail tous les points techniques sur lesquels il serait à désirer que l'accord soit fait pour que la méthode du séro-diagnostic tuberculeux soit employé partout, dans les mêmes conditions, avec le même succès qu'ont eu de nombreux observateurs.

1ère PARTIE.

ÉTAT DE LA QUESTION HISTORIQUE.

En 1898 M. Arloing fit connaître un procédé pour obtenir des cultures liquides homogènes du bacille de Koch, et montra le premier que ces cultures pouvaient être agglutinées par le sérum des hommes tuberculeux ou des animaux tuberculisés, créant ainsi le séro-diagnostic de la tuberculose.*

* *S. Arloing.*—"Sur l'obtention de cultures et d'émulsions homogènes du bacille de la tuberculose humaine en milieu liquide." 'C. R. de l'Académie des Sciences,' Paris, 9 mai 1898.

"Agglutination du bacille de la tuberculose vraie." 'C. R. de l'Académie des Sciences,' 16 mai 1898.

"Agglutination du bacille de Koch par le sérum sanguin des tuberculeux." 'Congr. de méd. interne,' Montpellier, 1898.

"Apparition dans le sérum sanguin, sous l'influence de produits chimiques, d'une matière capable d'agglutiner le bacille de la tuberculose vraie." 'C. R. de l'Académie des Sciences,' Paris, 31 mai 1898.

Digitized by Google

Certains perfectionnements dans l'obtention et le choix des cultures homogènes * et surtout les applications cliniques,† et la signification générale de la séro-réaction tuberculeuse ‡ furent étudiées par MM. Arloing et Paul Courmont dans de nombreuses publications.

Le séro-diagnostic de la tuberculose chez les bovidés a été ultérieurement étudié par M. Arloing.§

Les variations du pouvoir agglutinant des épanchements et des sérosités tuberculeuses ont été poursuivies par M. Paul Courmont dans différentes mémoires.‖

Depuis 1899, beaucoup d'auteurs se sont occupés de la question soit en France soit à l'étranger, et un certain nombre de points sont depuis longtemps hors de toutes contestations.

Actuellement, les faits suivants sont admis par tous les auteurs :—

1°. *On peut cultiver le bacille de Koch en cultures liquides homogènes.* Certains expérimentateurs, tels que Buard (Bordeaux), Bronstein (à Moscou), Carrière (à Lille), sont arrivés à ce résultat en partant de cultures solides différentes des nôtres. Tous ceux à qui nous avons envoyé de nos cultures homogènes les ont entretenues assez facilement.

2°. *Ces cultures homogènes sont très nettement agglutinées* à des taux variables par le sérum des hommes tuberculeux ou des animaux rendus tuberculeux. M. Beck et Mme Rabinowitsch, avec qui nous sommes en désaccord sur d'autres points, ont eux-mêmes reconnu qu'on peut élever le pouvoir agglutinant naturel du sang d'un animal en lui inoculant la tuberculose.

Quant aux applications pratiques et à la valeur clinique de la séro-réaction tuberculeuse, les opinions se partagent de la façon suivante.

Les uns confirment en tous points les résultats que nous avons obtenus

---

* *S. Arloing et Paul Courmont.*—"De l'obtention des cultures du bacille de Koch les plus propices à l'étude du phénomène de l'agglutination par le sérum sanguin des tuberculeux" ('C. R. de l'Académie des Sciences,' 19 septembre 1898).

† *S. Arloing et Paul Courmont.*—"Recherche et valeur clinique de l'agglutination du bacille de Koch" ('C. R. de l'Académie des Sciences,' 1898; 'Congr. pour l'étude de la tuberculose,' Paris, 1898). "Séro-diagnostic de la tuberculose" ('Congr. pour la lutte contre la tuberculose,' Berlin, mai 1899). "L'agglutination du bacille de Koch, séro-diagnostic" ('Zeitschr. f. Tuberk. und Heilstaettenwesen,' 1900. Band 1, Heft 1).

‡ *S. Arloing et Paul Courmont.*—"Des causes qui modifient le pouvoir agglutinant dans le sang des sujets expérimentalement tuberculeux" ('Journ. de physiol. et de pathol. générale,' 1900, No. 1). "Influence chez le chien d'une inoculation de bacilles de Koch très virulents sur le pouvoir agglutinant" ('Soc. de biol.,' 1900).

Voir: "Le séro-diagnostic de la tuberculose." 'Revue Générale,' 'Gazette des Hôpitaux,' 1er décembre 1900.

§ *S. Arloing.*—'Journal de Méd. Vétérinaire et de Zootechnie de l'École Vétérinaire de Lyon,' septembre 1900.

‖ *Paul Courmont.*—"Action des épanchements des séreuses sur le bacille de Koch en milieux liquides" ('Soc. de biol.,' 28 mai 1898). "Séro-diagnostic des épanchements tuberculeux" ('Presse méd.,' 11 juin 1898); ('Cong. de la tuberculose,' Paris, 1898). "Agglutination du bacille de Koch par les épanchements tuberculeux. Séro-diagnostic" ('Archiv. de méd. expérim. et Soc. de Biol.,' nov. 1900). Voir sur le même sujet: 'Th. de Feitu,' Lyon, 1900.

Digitized by Google

(Ferré, Mongour,* Buard,† Rothamel,‡ à Bordeaux ; Mosny,§ Ravaut à Paris, Carrière à Lille ‖ ; Bendix,¶ Romberg,** en Allemagne).

Le travail allemand le plus récent est celui de M. Romberg. Cet auteur, comme déjà Bendix, aboutit aux mêmes résultats que nous sur la fréquence de la séro-réaction chez les tuberculeux, sur la plus grande intensité de cette séro-réaction chez les tuberculeux à la 1ère ou à la 2e période, et par conséquent sur la valeur du séro-diagnostic pour déceler les cas de tuberculose peu avancée ou même latente. Le grand intérêt de la statistique de M. Romberg, qui porte sur 95 sujets, est que cet auteur est arrivé à ces résultats avec une technique différente de la nôtre, et sur laquelle nous reviendrons.

Quelques auteurs, confirmant les données essentielles du séro-diagnostic de la tuberculose, font seulement des réserves sur ses applications, la plupart parce qu'ils n'ont pu baser leur statistique sur un nombre suffisant de cas (Fraenkel †† de Halle, Knopf ‡‡ de New York, Ficker §§ de Leipzig, Bronstein ‖‖ de Moscou).

Enfin deux auteurs refusent toute valeur à la séro-réaction : M. Beck et Mme Rabinowitsch et M. Dieudonné.

Le travail de ce dernier est basé sur un trop petit nombre de cas pour qu'il puisse être opposé aux statistiques considérables de la plupart des auteurs.

Quant aux objections de M. Beck et Mme Rabinowitsch ¶¶ nous les avons déjà longuement réfutées.*** Les idées de ces auteurs sur l'importance qu'on doit attacher à l'intensité de la séro-réaction en générale, sur le phénomène de l'agglutination en lui-même, leur sont trop spéciale pour être admises par quiconque est au courant de la pratique du séro-diagnostic en générale. Les

---

* *Mongour et Buard.*—('Soc. de biol.,' 11 juin et 26 dec. 1899. 'Journ. de méd. de Bordeaux,' 17 dec. 1898, et 9 juillet 1899. *Mongour.*—"Rapport à la Réunion médico-chirurgicale des hôpitaux de Bordeaux," 1899).

† *Buard.*—"Séro-réaction tuberculeuse. Cultures du bacille agglutinable. Étude spéciale chez l'enfant." ('Th. de Bordeaux,' 1900. 'Journ. de Physiol. et Pathol.' sept. 1900). "Séro-réaction tuberculeuse" ('Gaz. hebd. des sc. méd. de Bordeaux,' sept. 1900).

‡ *Rothamel.*—"Agglutination du bacille de la tuberculose, principalement chez les tuberculeux cachectiques" ('Th. de Bordeaux,' nov. 1899).

§ *Mosny.*—'Congrès de Paris,' 1900.

‖ *Carrière.*—"Le séro-diagnostic de la tuberculose" ('Société de Biologie,' 6 juillet 1901).

¶ *Bendix.*—"Zur séro-diagnose der Tuberkulose" ('Deutsch. med. Wochen.,' 5 avril 1900, p. 224).

** *Romberg.*—"Zur séro-diagnose der Tuberkulose" ('Deutsch. med. Wochen.,' 1901, Nos. 18–19).

†† *Fraenkel.*—"Séro-diagnostic de la tuberculose d'après les recherches de S. Arloing et P. Courmont" ('Hygien. Rundschau,' 10 juillet 1900).

‡‡ *Knopf.*—"Early recognition of pulm. tubercul." ('Journ. of the Amer. Med. Assoc.,' Dec., 1899).

§§ *Ficker.*—"Ueber die Serumreaction bei Tuberkulose" ('Zeitschrift für Tuberculos.,' B. 1, Heft 4).

‖‖ *Bronstein.*—('Soc. de pédiatrie de Moscou,' 1901, et Wratch, 1901).

¶¶ *Beck et Rabinowitsch.*—('Deutsche med. Wochen.,' juin 1900, No. 25).

*** *S. Arloing et P. Courmont.*—"Sur la valeur de la séro-réaction pour le diagnostic précoce de la tuberculose" ('Presse méd.,' 1er sept. 1900, No. 73, et 'Deutsch. med. Wochen.,' nov. 1900, Bd. 48).

Digitized by Google

divergences de ces auteurs doivent être probablement mises sur le compte de conditions différentes d'observation et de technique.

2e PARTIE.

TECHNIQUE DE LA RECHERCHE DE LA SÉRO-RÉACTION DANS LA TUBERCULOSE. DIFFICULTÉS ET CAUSES D'ERREUR.

La technique de l'entretien des cultures homogènes et de la recherche de la séro-réaction présente quelques difficultés sérieuses pour les débutants parce qu'elle exige des soins et des précautions que ne comporte pas le séro-diagnostic de la fièvre typhoïde par exemple.

C'est là sans doute ce qui a occasionné les échecs de quelques uns et les réserves de certains auteurs au sujet de la question qui nous occupe.

Mais ces difficultés peuvent être facilement vaincues avec un peu de persévérance et quelques précautions dans un laboratoire bien organisé.

Nous allons exposer tout d'abord les deux sources principales de difficultés théoriques et pratiques; nous développerons ensuite tout au long la technique de la méthode dans tous ses détails et avec les perfectionnements que nous y avons apportés.

I. Principales Difficultés et Causes d'Erreur.

Nous les avons déjà signalées souvent: nous allons résumer ici les points principaux.

Les causes d'erreur dans l'application du séro-diagnostic de la tuberculose sont de deux sortes: les unes tenant à la nature même de la maladie, les autres aux propriétés des cultures.

A. *Causes d'erreur tenant à la nature même des localisations et de l'évolution de la tuberculose.*

Dans certaines maladies à marche cyclique et typique comme la fièvre typhoïde le diagnostic clinique, dans la plupart des cas, finit par être posé avec certitude, et il est facile de comparer les données de la clinique à celle du séro-diagnostic. Mais pour la tuberculose, dont les localisations sont multiples, dont l'évolution peut être très longue, se présenter sous les aspects les plus variables, et même coincider avec un état de santé normal en apparence au moins pendant quelque temps, pour la tuberculose les difficultés de diagnostic clinique sont souvent très grandes, surtout au début. De telle sorte que dans beaucoup de cas un séro-diagnostic à juste titre positif peut être en désaccord avec les données de la clinique, surtout lorsque celles-ci sont superficielles et s'adressent à une maladie intercurrente sans viser spécialement la tuberculose. C'est ainsi que nous avons vu très souvent les données du séro-diagnostic confirmées par l'autopsie alors qu'elles étaient en désaccord avec la clinique pendant la vie du malade.

Digitized by Google

C'est pour cela que les expérimentateurs qui pratiquent la séro-réaction tuberculeuse avec du sang de malades dont ils n'ont pas soit l'examen clinique complet, soit le protocole d'autopsie, ne peuvent tirer de leur recherche aucune conclusion valable. Comme d'autre part, certains tuberculeux avérés (les plus malades ordinairement) peuvent ne pas donner la séro-réaction, comme nous l'avons depuis longtemps établi, et qu'enfin, des sujets tuberculeux en réalité, mais sains en apparence, peuvent donner une réaction très positive, certains auteurs ont conclu à tort de cette apparente contradiction à la faillite du séro-diagnostic de la tuberculose.

Pour faire avec fruit des recherches démonstratives sur cette question il faut pouvoir : d'une part, étudier la séro-réaction au laboratoire avec toutes les précautions voulues, et, d'autre part, établir un diagnostic clinique aussi complet que possible avec toutes les ressources actuelles (examen clinique, analyse des crachats, emploi de la tuberculine s'il y a lieu, etc.). En réalité une statistique indiscutable ne devrait se baser que sur des cas ou l'autopsie complète aurait pu être faite, avec examen anatomo-pathologique et bactériologique lorsqu'il serait nécessaire. C'est ce qu'a fait M. Arloing pour la tuberculose des bovidés.* Comme cet idéal n'est pas toujours possible pour les statistiques humaines, il faut s'en rapprocher en ne comparant les résultats de la séro-réaction à ceux de la clinique que lorsque ceux-ci seront sûrement établis.

B. *Causes d'erreur tenant au développement des cultures.*—Ces causes d'erreur longuement exposées ailleurs par nous † ont été récemment développées et même exagérées par M. Ficker.‡

Cet auteur montre bien d'ailleurs, ainsi que M. Romberg, que c'est là qu'il faut chercher la raison des divergences inexpliquées de quelques expérimentateurs. Mais ils vont trop loin en disant que ces difficultés rendent le procédé inapplicable parce que certains n'ont pas su en triompher.

Elles peuvent d'ailleurs se résumer en trois propositions :—1°. *Il est réellement difficile et délicat de transformer en culture liquide homogène un échantillon ordinaire de bacille de Koch.* Actuellement cette difficulté n'en est plus une, puisque nous envoyons à qui nous le demande notre échantillon de bacille en culture liquide. Il serait même préférable que les auteurs qui veulent essayer la méthode le fassent avec le bacille que nous employons.

2°. *Les cultures homogènes présentent une grande variabilité dans leur aptitude à se laisser agglutiner*, à cause de leur richesse variable selon les conditions de développement, leur âge, etc. Celle-ci peut être surmontée par l'usage d'une technique toujours rigoureusement la même pour un même observateur. L'emploi d'un *sérum-étalon* et l'usage de *cultures formolées* aplanissent la plupart de ces difficultés.

3°. *Le pouvoir agglutinant des sérums humains tuberculeux étant peu élevé* (il dépasse rarement 1 pour 20) il faut tenir compte de différences souvent

* *S. Arloing*, loc. sit.

† Voir : 'Gazette des hôp.,' 1er décembre 1900.

‡ *Ficker*, loc. cit.

Digitized by Google

minimes pour apprécier exactement une séro-réaction. Sur ce dernier point, nous verrons plus loin la façon de résoudre le problème (emploi du sérum-étalon et appréciation de la réaction a l'œil nu).

Il est bien certain d'ailleurs que ces deux derniers points suffisent à expliquer les divergences de quelques uns. Il suffit que de plusieurs auteurs, l'un ne tienne pas un compte suffisant des précautions à prendre pour éviter la variabilité des cultures, ou bien n'apprécie pas d'après la même méthode les degrés et les limites de l'agglutination, pour que les résultats de cet auteur diffèrent de ceux qui se sont placés dans des conditions plus rigoureuses et identiques. Aussi nous allons insister sur les précautions à prendre pour éviter toute cause d'erreur.

II. Obtention des Cultures Homogènes de Bacilles de Koch en Milieu Liquide.

Le résultat s'obtient en deux temps : dans un premier, on tâche d'arriver à des cultures sur pommes de terre dont les colonies rapprochées, luxuriantes, ont un aspect gras et brillant ; dans un second, on s'efforce d'acclimater ces colonies dans des milieux liquides. (Voir pour les détails : S. Arloing, 'Ac. des Sciences,' 9 mai 1898.)

La principale condition pour conserver ensuite l'homogénéité dans des générations successives est *l'agitation des matras*, qui combat la tendance du bacille à végéter en grumeaux ou en voile. Ajoutons, toutefois, que cette tendance diminue à mesure que les générations deviennent plus nombreuses. Avec le temps, nous sommes même parvenus à faire végéter facilement les bacilles dans du bouillon non glycériné.

Si les cultures vieillissent au repos, quelques colonies montent et se développent à la surface du bouillon. On distingue alors trois étages dans le matras : au fond, une couche de bacilles isolés ou réunis en grumeaux très fins ; au milieu, le bouillon limpide et transparent, en haut, une couche de bacilles isolés comme dans un mycoderme.

Les cultures homogènes s'éloignent du type classique. Ainsi, portées sous le microscope, elles présentent à coté de petits amas une majorité de bacilles isolés jouissants d'une certaine mobilité ; transportées sur milieu solide elles donnent au bout de quelques jours un voile crémeux, blanchâtre, uniforme ou bien gaufré. Le transport sur gélose simple et sur gélose glycérinée est un des meilleurs moyens de se rendre compte de la pureté d'une culture. Sur gélose simple elles ne poussent pas, le plus souvent, ou bien seulement au bout d'un temps assez long. Sur gélose glycerinée on n'obtient des colonies qu'au bout de trois ou quatre jours ; cela suffit d'ordinaire pour vérifier la pureté de la culture.

Une autre conséquence importante de cette modification retentit sur les affinités du bacille de Koch pour les matières colorantes, surtout lorsqu'on s'éloigne de la culture souche. Prenons comme type : 1° Une culture homogène de 1ère ou 2e génération provenant de la culture sur pommes de terre : 2° Une culture jaune de dixième ou vingtième génération, provenant également

Digitized by Google

d'une culture sur pomme de terre, mais dont les générations successives auront été rapprochées les unes des autres; les bacilles du second type se laisseront facilement décolorer par les acides, tandis que ceux du premier conserveront plus ou moins complètement la propriété de résister aux décolorants. Par contre, si nous laissons vieillir la culture du second type, nous voyons que le bacille récupère peu à peu ses caractères classiques; il résiste aux décolorants (méthode de Ziehl ou d'Häuser) alors qu'il n'avait pas ce caractère pendant les premiers jours de son développement.*

L'expérimentateur qui emploie ces cultures doit connaître la variabilité de leurs caractères pour ne pas se laisser tromper ni prendre pour des cultures impures celles où les bacilles auraient perdu une partie de leurs propriétés classiques. Le retour de ces dernières à la normale par vieillissement sera toujours un critérium à employer.†

La variabilité ne porte pas seulement sur la résistance aux décolorants, et sur la virulence, elle se manifeste encore dans l'aptitude des cultures à se laisser agglutiner.

III. Obtention des Cultures les plus propices à l'Agglutination.

Dans la recherche des cultures les plus propices à l'agglutination il faut se préoccuper des conditions suivantes.

Le point capital qui rend le séro-diagnostic plus difficile dans la tuberculose que dans les autres maladies est le suivant: Les cultures homogènes de bacille de Koch présentent une variabilité considérable dans leur aptitude à se laisser agglutiner. Cette variabilité dépend surtout d'un facteur, leur richesse en bacilles et en matière agglutinable.

Une culture jeune et pauvre en bacilles sera facilement agglutinable; une culture âgée sera trop riche, et trop "dure" à l'agglutination, et donnera à tort des résultats négatifs. Pour un même sérum, la résistance de deux cultures de richesse inégale sera souvent très différente. Cette aptitude à se laisser agglutiner et par conséquent cette richesse des cultures dépendra naturellement des conditions suivantes.

(*a.*) *Le milieu.*—Nous avons indiqué la formule du milieu le plus convenable: c'est le bouillon de bœuf ou de veau peptoné à 1 ou 2 pour cent et glycériné à 6 pour cent. Il est facile de le préparer dans les conditions toujours identiques. Nous employons la peptone Whitte ou Chapoteaux et de la glycérine neutre. La quantité et la qualité de la glycérine et de la peptone, la réaction du milieu (les milieux acides sont les plus souvent dysgénésiques, comme cela arrive parfois avec les peptones de mauvaise qualité), la concentration du bouillon, ont leur importance. Cependant M. Ficker a peut-être

* M. Marmoreck ('Congrès de Paris,' 1900) a publié des résultats analogues sur la perte des propriétés colorantes ordinaires des cultures de bacille de Koch lorsqu'elles sont jeunes.

† Notre bacille est peu virulent; il peut cependant donner la tuberculose épiploïque chez le lapin par injection dans le péritoine. Il peut donner, irrégulièrement chez le cobaye, une tuberculisation généralisée.

Digitized by Google

exagéré leur influence. Un même observateur peut bien se mettre toujours dans des conditions favorables et toujours identiques.

(*b.*) *La température de l'étuve.*—Il est indiqué de cultiver le bacille toujours à la même température, 38° ou 39°.

(*c.*) *L'agitation des cultures.*—Il faut agiter régulièrement les ballons au moins une fois par jour, toujours de la même façon.

(*d.*) *La forme des ballons.*—Il est bon d'employer toujours le même modèle des ballons. Ceux-ci seront de préférence à fond plat, avec une couche de bouillon de peu d'épaisseur.

(*e.*) *La quantité de culture ensemencée.*—Il va sans dire qu'une culture largement ensemencée sera plus rapidement riche en bacilles. Il faut donc ensemencer toujours à peu près la même quantité pour un volume donné de bouillon.

(*f.*) *L'âge de la culture mère.*—Cette condition a de l'importance surtout parce que la semence sera plus ou moins riche en bacilles selon son âge. Ces deux derniers facteurs ont été de nouveau mis en relief par M. Ficker.

(*g.*) *L'âge auquel on emploie la culture pour l'agglutination.*—D'après ce que nous avons dit, étant donné l'accroissement un peu lent mais progressif des cultures, leur richesse en bacilles et leur difficulté à se laisser agglutiner augmentent avec leur âge.

Mais, d'autre part, comme les autres facteurs signalés ont aussi leur influence sur le développement plus ou moins rapide des cultures de même âge, celles-ci peuvent être de richesse différente si les autres conditions ont varié, et réciproquement des cultures d'âge différent peuvent avoir les mêmes propriétés.

En règle générale, il vaut mieux se servir de cultures qui, moyennement ensemencées avec une culture riche âgée de quatre à huit semaines, ont poussé assez rapidement. Celles dont la végétation a été lente et pénible sont souvent remplies d'onnas ou "dures" à l'agglutination.

Les cultures riches, âgées de huit à douze jours, nous ont paru les plus favorables à la réaction agglutinante. Lorsqu'elles datent de plus de quinze jours, la clarification est difficile à obtenir d'une façon complète avec la même dose de sérum, à moins qu'il ne s'agisse d'un sérum particulièrement agglutinant. Un caractère d'une certaine valeur est tiré de la viscosité; il faut que celle-ci soit assez grande pour que la culture mousse bien par mélange avec l'air.

Enfin il faut savoir que les cultures liquides de bacilles de Koch, d'abord péniblement et artificiellement développées, poussent de mieux en mieux, et plus rapidement à mesure qu'on les réensemence fréquemment. C'est un phénomène que nous avons nettement constaté depuis deux ans; nos cultures poussent mieux et plus vite actuellement qu'en 1898. De telle sorte que nous obtenons en un temps plus court (huit à douze jours) des cultures propices à l'agglutination et avec lesquelles nous observons le phénomène en quelques heures (de une à cinq) sans être obligés d'attendre dix à vingt heures comme dans les premiers temps. M. Buard a constaté des faits analogues. En pratique il emploie des cultures plus jeunes que nous et observe l'agglutination en un temps plus court.

Digitized by Google

En résumé, la condition essentielle est de n'employer que des cultures toujours semblables à elles-mêmes comme richesse et comme aptitude à l'agglutination. Ce résultat sera obtenu, sans très grandes difficultés, si l'on se met toujours dans les mêmes conditions, avec une technique toujours identique pour un même observateur. Certaines des conditions que nous avons indiqués pourront en effet varier quelque peu avec les différents expérimentateurs; l'un emploiera des cultures plus jeunes, lorsque celles-ci plus richement ensemencées seront plus vite parvenues au point voulu, un autre se servira de cultures un peu plus âgées lorsque, dans les conditions où il opère, elles se seront développés plus lentement.

On comprend qu'on ne puisse fixer des règles mathématiques pour un tel objet.

Mais nous pouvons dire par expérience, que dans un laboratoire bien organisé où l'on entretient continuellement ces cultures, l'ensemble des conditions nécessaires pour qu'elles aient les propriétés voulues n'est pas très difficile à réaliser et ne demande qu'un peu de constance et de patience.

Nous répétons que l'habitude est encore le meilleur guide, et que "l'application de la méthode en clinique réclame une période de tâtonnement et d'éducation pour chaque observateur, qui devra surtout s'habituer, avant de passer aux applications, à bien connaître les caractères de sa culture, la rapidité de son développement et son agglutinabilité à telle ou telle période."

IV.—Emploi du Sérum-Étalon.

Il est un moyen très simple et très pratique de se mettre à l'abri des causes d'erreur et de juger soit du degré d'agglutinabilité d'une culture soit de la valeur à accorder à une réaction plus ou moins complète dans un cas donné; c'est l'emploi d'un serum dont le pouvoir agglutinant est connu une fois pour toutes et qui sert de commune mesure dans tous les essais de séro-réaction

Il est facile de recueillir aseptiquement et de conserver (de préférence dans un endroit frais) * un sérum d'animal tuberculisé, ou une sérosité de pleurésie tuberculeuse qui donne une agglutination nette. Par tâtonnements, en comparant le même jour, avec la même culture, dans les mêmes conditions, son pouvoir agglutinant, avec celui d'un certain nombre d'autres sérums tuberculeux, on fixe une fois pour toutes son pouvoir agglutinant. On peut aussi, et pour plus de facilité, comparer ce sérum à celui employé par un autre expérimentateur.

Dès lors, chaque fois que l'on emploiera une nouvelle culture, on fera une agglutination témoin avec le sérum étalon. Seules les cultures donnant le degré voulu d'agglutination seront employées pour les séro-diagnostics et les résultats seront rapportés à celui du sérum étalon servant de commune mesure.

* Un sérum peut ainsi conserver ses propriétés plusieurs mois, s'il est aseptique, à l'abri d'une trop grande chaleur.

Digitized by Google

Si la culture paraît trop jeune, on la remet à l'étuve pour l'essayer de nouveau plus tard. Si elle est trop riche et trop "dure," on peut la diluer avec du bouillon ordinaire jusqu'à ce que le mélange donne le taux voulu d'agglutination avec le sérum-étalon. On juge assez facilement à la vue du degré de dilution auquel on doit s'arrêter.

L'habitude finit d'ailleurs par guider tellement dans l'appréciation des qualités d'une culture (trouble, viscosité, rapidité de développement, etc.) que nous arrivons facilement à employer ou à rejeter une culture donnée, rien que pour son aspect extérieur, mais nous contrôlons toujours ces données subjectives par l'emploi d'un sérum-étalon. Il est bien rare que ce critérium ne vienne pas confirmer l'opinion que nous avions d'une culture.

On voit donc qu'il ne faut rien exagérer, et que si la pratique du séro-diagnostic est délicate et demande des soins constants, elle est à la portée de tout expérimentateur consciencieux opérant dans un laboratoire bien outillé.

Nous croyons donc plus que jamais, qu'il ne faut pas attacher trop grande importance à quelques difficultés de pratique, et conclure, comme l'ont fait certains, à l'impossibilité d'user avec fruit de la technique que nous avons indiquée, et dont bien d'autres se sont servi heureusement.

Nous allons d'ailleurs indiquer un moyen de simplifier encore la méthode.

V. Emploi des Cultures Formolées.

La principale difficulté avons-nous dit est précisément cette variabilité des cultures.

En effet lorsqu'une culture, obtenue par les soins que nous avons indiqués, est parvenue au point voulu, elle n'y demeure pas longtemps, puisque, continuant à végéter, elle devient de plus en plus riche et de plus en plus difficile à agglutiner. De telle sorte qu'il faut s'addresser à une nouvelle culture entretenue avec les mêmes soins et contrôlée par les mêmes procédés.

Il était donc indiqué de rechercher un moyen de préparer la culture convenable et de l'arrêter dans sa végétation au point voulu pour qu'elle conserve ses propriétés.

On aurait ainsi un produit que l'on pourrait conserver et employer à volonté selon les besoins de chaque jour.

Depuis longtemps, nous employons le procédé suivant: la conservation de la culture au froid; nous avions aussi de temps en temps employé des cultures fixées par le formol.

Tout récemment M. Romberg * a employé pour le séro-diagnostic une émulsion de bacilles préparée par M. Behring. Ce dernier avait constaté l'agglutination d'une telle préparation par le sérum d'animaux tuberculeux.

Le procédé de MM. Behring et Romberg ne nous paraît pas être le perfectionnement désiré.

En effet: 1°. La préparation de leur émulsion semble (autant qu'on en

* *Romberg*, loc. cit: "Zur Serumdiagnose des Tuberkuloses." 'Deutsch. med. Woch.,' 1901, No. 18–19.

Digitized by Google

peut juger par le travail de Romberg) assez compliquée à préparer, puisqu'il faudrait faire agir de la lessive de soude pendant plusieurs jours sur des bacilles préalablement séchés. D'ailleurs il est probable que la culture des bacilles en question demande le même manuel opératoire que nos cultures liquides.

2°. La recherche de l'agglutination des corpuscules contenus dans cette émulsion paraît longue. Elle nécessite plusieurs centimètres cubes de sérum, deux tubes témoins (tube de la solution seule et du sérum seul) et l'auteur conseille aussi l'emploi d'une réaction comparative avec un sérum sûrement agglutinant.

Enfin il faudrait au moins 19 à 24 heures, parfois deux jours, pour que la réaction soit complète.

Notre méthode qui n'emploie que quelques gouttes de sérum,* quelques centimètres cubes de culture, de tout petits tubes n'occupant que peu de volume, et qui surtout peut donner le résultat en quelques heures (parfois une heure, ordinairement de quatre à cinq heures), nous semble de tous points préférable comme simplicité et rapidité.

Nous sommes heureux d'ailleurs que ce soit avec un tel procédé différent du nôtre dans ses détails que M. Romberg soit arrivé dans son remarquable travail à confirmer nos résultats et à conclure à la grande valeur du séro-diagnostic.

En tout cas voici les procédés que nous proposons après les avoir expérimentés longuement.

A. *Conservations des cultures au froid.*

Nous avons souvent conservé des cultures propices à l'agglutination en les plaçant dans un endroit froid où la végétation ne puisse plus se faire. Elles peuvent ainsi se maintenir quelques temps avec les mêmes propriétés. Quand un dépôt naturel s'est formé en abondance il suffit de l'agiter et de le mettre en suspension avant de se servir de la culture. Mais, à la rigueur, des cultures ainsi conservées pourraient se contaminer, ou bien se modifier en été; lorsqu'on les emploie à une température élevée. En tout cas elles ne pourraient pas facilement s'envoyer au loin sans se modifier quelque peu.

B. *Conservation des cultures par le formol.*

Depuis longtemps M. Widal † a démontré que les bacilles d'Eberth tués par le formol (1 goutte pour 150) se laissent agglutiner par le sérum des typhiques.‡

---

* Avec 1 cc. à 2 cc. de sang on a largement la quantité de sérum suffisante dans notre méthode. Une si petite quantité se prend facilement au bout des doigts et ne nécessite pas une ponction veineuse ou une ventouse scarifiée comme le procédé de Romberg.

† *Widal.*—"Agglutination des bacilles morts." 'Soc. de Biol.,' 1897, 'Annales de l'Inst. Pasteur,' mai 1897.

‡ Nous avons essayé des cultures de tuberculose depuis par la chaleur, mais les résultats ont été peu satisfaisantes.

Digitized by Google

Nous avions fait la même constatation il y a longtemps pour le bacille de Koch. Depuis quelques mois, nous employons ce moyen pour conserver nos cultures agglutinables. Une faible dose de formol du commerce (1 ou 2 de formol pour 200 de culture) ne semble pas modifier l'agglutinabilité de ces cultures, et elles se conservent d'un trouble bien homogène pendant quelque temps. Comme il se fait un dépôt de bacilles, il faut l'agiter fortement pour restituer au liquide son trouble primitif. A cette dose de 1 ou 2 pour cent les cultures paraissent tuées; dans tous les cas, réensemencés en bouillon elles ne poussent pas, et la plupart des germes vulgaires ne risquent pas non plus de contaminer le milieu.

Le manuel opératoire est donc le suivant :

1° Faire dans des ballons à fond plat contenant une couche peu épaisse de bouillon, des cultures de bacille de Koch homogènes en observant les précautions usuelles.

2° Lorsque la culture paraît à point, on essaie le degré d'agglutinabilité des différents ballons avec un sérum-étalon.

Les ballons trop pauvres sont remis à l'étuve, les ballons trop riches sont dilués avec du bouillon jusqu'au point voulu, ce qui se fait facilement avec un peu d'habitude.

On mélange les cultures qui donnent ainsi le taux voulu d'agglutination par des sérums connus, on ajoute au mélange la dose indiquée de formol, et on répartit la culture totale bien agitée dans de petits flacons dont on se servira au fur et à mesure des besoins. Il sera bon d'ailleurs pour plus de sureté, lorsqu'on commence à user de chacun de ces petits ballons, de l'éprouver avec les sérums-étalon ou même plusieurs sérums connus.

On peut avoir ainsi une culture liquide, normale, totale, ayant subi le minimum de manipulations et dont le degré d'agglutinabilité est connue et fixe pour quelques temps. De telles cultures peuvent être maniées facilement puisque les bacilles sont rendus inactifs, elles peuvent être expédiées au loin aux expérimentateurs qui veulent en user. Le même degré d'agglutinabilité des cultures formolées ne se conserve pas indéfiniment; il baisse au bout d'une dizaine de jours. Il est donc prudent de ne pas se servir de cultures formolées depuis plus de 15 jours environ.

De cette façon, et sans modifier aucunement le manuel opératoire très simple et très pratique, du séro-diagnostic, on peut très facilement parer à la difficulté de la variabilité des cultures.

VI. Technique du Séro-diagnostic, Manière d'apprécier l'Agglutination.

Les détails que nous allons donner visent surtout la recherche du pouvoir agglutinant du sérum sanguin des hommes soupçonnés de tuberculose.

A. *Technique générale.*—(*a.*) *Le sérum* doit être préféré au sang total, dont les globules peuvent masquer le dépôt; le sérum incolore, au sérum coloré par l'hémoglobine; le sérum frais, à celui qui aurait pu être souillé par des germes vulgaires. On l'obtiendra de la rétraction spontanée du sang qui s'est écoulé

Digitized by Google

dans de petits tubes ad hoc, à la suite d'une piqûre de lancette faite à la pulpe d'un doigt dont la surface a été préalablement aseptisée. Il suffit en général de quelques gouttes de sérum clair et par conséquent d'1 ou 2 cc. de sang, ce qu'il est très facile d'obtenir par piqûre du doigt. À défaut de la rétraction spontanée du caillot, on a recours à l'écrasement de coagulum, puis à l'action de la force centrifuge pour bien séparer le sérum de la partie solide.*

(*b.*) *Les petits tubes* stérilisés dans lesquels on fait la réaction auront un petit diamètre pour que la colonne liquide soit d'une certaine hauteur (les nôtres ont 7 m/m de diamètre). Une fois le mélange bien accompli à leur intérieur par leur agitation, on les inclinera à 45 degrés afin que les grumeaux se déposent plus facilement, au fur et à mesure de leur formation, sur une certaine longueur des tubes. Autant que possible on ne les agitera pas avant que la réaction ne soit faite.

(*c.*) *Les dilutions.* Avec chaque sérum on prépare trois ou quatre mélanges de titre différent, à 1/5, 1/10, 1/15 et 1/20 ; c'est-à-dire à une goutte de sérum pour cinq de culture et ainsi de suite.

L'agglutination n'a pas ou n'a que peu de signification pratique dans un mélange dont le titre est supérieur à 1/5 ; ordinairement, elle fait défaut au-dessous de 1/20. Cette échelle de trois tubes facilite l'observation dans les cas limites. Il va sans dire qu'on pourrait employer un plus grand nombre de tubes : la dilution à 1/15 sera employée avec fruit.

(*d.*) *Le temps* au bout duquel la séro-agglutination est complète, ordinairement plus long que dans la séro-agglutination typhoïde, varie d'ailleurs beaucoup suivant les sérums.

Les modifications les plus importantes se passent *dans les premières heures.* Les modifications qui se produiraient au delà sont de plus en plus négligeables.

(*e.*) *Le sérum-étalon.* Chaque fois que nous faisons un séro-diagnostic nous pratiquons par comparaison la réaction sur la même culture avec le sérum-étalon, comme il a été indiqué plus haut.

B. *Manière d'apprécier la limite de l'agglutination.*

Lorsqu'un sérum agglutinant est mis en contact, dans les conditions et les proportions que nous avons indiquées, avec une culture homogène convenablement préparée, on observe les phénomènes suivants, comme dans toute agglutination. On voit, en temps variable, se former de petits grains qui se réunissent en flocons et finissent par se précipiter en dépôt au fond du tube, laissant au-dessus de ce dépôt un liquide clarifié, parfois absolument limpide orsque la réaction se fait au maximum.

Ces phénomènes peuvent se constater soit à l'œil nu soit au microscope, et nous avons par conséquent les différents éléments suivants d'appréciation.

---

* M. Buard conseille deux variantes du procédé : l'emploi du sang desséché, et le mélange à parties égales de sérum et de cultures en examinant immédiatement au microscope. Nous croyons que ces procédés ne doivent être employés que par des observateurs très exercés et rompus au maniement de la séro-réaction. Nous ne les conseillerions pas d'une façon générale et surtout pour les débutants.

Digitized by Google

1°. *A l'œil nu :* d'abord l'apparition des flocons, puis la clarification avec formation du dépôt.

Ces deux phénomènes qui se succèdent * présentent trois degrés :

1^er^ *degré :* formation lente de très petits flocons, de grains plutôt, à peine visibles à l'œil nu, ne se déposant que partiellement, de sorte qu'il n'y a qu'un dépôt faible ou nul et pas de clarification du mélange, qui reste trouble. Dans ces cas, au microscope, on voit des amas très lâches, à bacilles très éloignés les uns des autres, et de très nombreux bacilles isolés.

C'est là ce que nous appelons une *ébauche de réaction agglutinante*, et dont nous ne tenons pas compte pour une séro-diagnostic, si ce n'est à titre d'indication.

2^me^ *degré :* formation peu rapide de flocons peu volumineux, dépôt consécutif de moyenne abondance, surmonté d'une colonne liquide partiellement clarifiée mais encore manifestement louche.

Au microscope on voit des amas de moyenne grosseur, mais encore beaucoup de bacilles isolés.

C'est ce que nous appelons une *réaction incomplète ;* nous la signalons souvent par le signe ±.

3^me^ *degré :* formation rapide de flocons volumineux qui peuvent soit se précipiter isolément, soit se réunir en une sorte de coagulum rétracté en boule ou à bords déchiquetés ; dépôt abondant ; clarification de la colonne liquide qui surmonte le dépôt :

Au microscope on ne voit que de très gros amas et pas de bacilles libres (sauf quelques-uns que la manipulation aura détaché des amas).

C'est ce que nous appelons la *réaction positive complète*, nous la marquons du signe + ou $\frac{+}{+}$ suivant sa rapidité et son intensité.

Il va sans dire qu'il y a entre ces degrés tous les intermédiaires, mais il est facile avec un peu d'habitude de ranger les réactions observées dans une de ces catégories en indiquant les nuances par l'association des signes $\frac{\pm}{\pm}$ $\frac{+}{\pm}$ $\frac{+}{+}$ etc.

2°. *Au microscope*, les phénomènes observés sont sans doute, comme nous venons de le dire, en rapport avec les phénomènes macroscopiques et, pour mieux dire, ils n'en sont que l'amplification. Avant que l'œil puisse apercevoir le flocon dans le tube, le microscope peut montrer que les amas sont en voie de formation. Si bien que l'on pourrait avoir tendance à n'employer que la réaction microscopique comme cela se pratique couramment pour la fièvre typhoïde par exemple. Nous croyons que si une telle pratique donne de bons résultats pour l'agglutination de bacilles d'Eberth, elle en donnerait de moins bons pour l'agglutination du bacille de Koch. Nos raisons en sont les suivantes. Il arrive très fréquemment qu'une culture, même bien entretenue, de bacille de Koch en milieu liquide, présente un grand

* Il faut savoir que, parfois, précisément avec des sérums très agglutinants, le coagulum total formé par l'agglutination reste en suspension et peut gêner l'appréciation de la clarification.

Digitized by Google

nombre de petits amas qui gênent l'observateur dans l'appréciation de l'agglutination, même par comparaison avec une goutte de la culture témoin.

De plus, les amas de bacilles tuberculeux agglutinés sont assez fragiles, une agitation trop forte avec la pipette ou toute autre cause de ce genre peut les dissocier et donner des bacilles isolés en plus ou moins grand nombre. Enfin il est certain qu'une réaction sera toujours plus facilement et plus vite observée à l'œil nu qu'au microscope.

On objectera peut-être que le microscope donne une plus grande finesse de résultats, en montrant par exemple, dans les cas d'ébauche de séro-réaction, des amas que l'œil nu ne saurait percevoir dans le liquide resté trouble. Nous répondons que c'est là précisément un des inconvénients de l'emploi du microscope pour fixer, dans le cas qui nous occupe, la limite d'une agglutination tuberculeuse. En pratique, il ne faut tenir compte que des résultats certains, des réactions bien nettes et l'emploi (ou du moins l'emploi exclusif) du microscope exposerait à trop s'embarrasser dans l'appréciation des résultats, de ces réactions douteuses.

C'est précisément parce que la technique du séro-diagnostic de la tuberculose est délicate, parce que l'échelle du pouvoir agglutinant du sérum des tuberculeux est limitée, qu'il ne faut tenir compte que des résultats bien nets.

C'est pour cela que nous attachons, en pratique, l'importance la plus grande à la réaction macroscopique, parce que, lorsqu'elle est nette il n'y a pas de doute possible, et que dans ces cas le microscope donnerait la même réponse, alors que l'inverse n'est pas vrai. Le chiffre d'agglutination peut être parfois moins élevé à l'œil nu qu'au microscope, de telle sorte que notre manuel opératoire nous rend plus sévère dans l'acceptation d'un séro-diagnostic positif, mais cela vaut mieux en pratique.

En résumé :

1°. Il y a deux moyens de constater l'agglutination : l'œil nu et le microscope. *Nous donnons la préférence à l'examen de la réaction macroscopique* pour la pratique du séro-diagnostic tuberculeux.*

2°. Une séro-réaction tuberculeuse est *complète* (gros flocons et dépôt et clarification), *incomplète* (flocons moyens avec ou sans dépôt, éclaircissement sans clarification,) ou *ébauchée* (très petits grains, parfois seulement amas microscopiques, pas d'éclaircissement du liquide).†

Nous ne tenons compte des réactions incomplètes qu'à titre d'indication.

Enfin il va sans dire que les réactions positives complètes n'ont de valeur qu'à partir du titre de dilution où le sérum normal de l'espèce considérée ne donne pas cette agglutination.

Par conséquent, pour qu'un séro-diagnostic soit positif à notre point de vue, il faut deux choses : d'abord que la séro-réaction soit positive et complète,

---

* Tout ceci ne veut pas dire qu'il ne faille employer constamment le microscope pour juger de la pureté d'une culture, de l'état des bacilles dans celle-ci, etc.

† Une réaction peut être dite positive complète ou incomplète, à des titres divers. Par exemple, le pouvoir d'un sérum tuberculeux qui agglutinera complètement à 1/10, incomplètement à 1/15, et pas du tout à 1/20, s'exprimera + 10 ± 15.

Digitized by Google

comme nous l'avons indiquée, et ensuite qu'au titre de dilution du sérum où cette réaction a été faite le sérum normal de l'espèce considérée ne soit pas agglutinant.

On sait en effet que le sérum de la plupart des espèces animales agglutine normalement le bacille de Koch à des degrés divers selon l'espèce considérée : par exemple le sérum de cheval à 1 p. 15 et au-delà, le sérum de vache normale agglutine complètement à 1 p. 5 et incomplètement à 1 p. 10 . . . etc. . .

C'est pour cela que nous demandons que dans la pratique du séro-diagnostic on tienne compte seulement des réactions complètes à partir d'un certain degré de dilution dont le taux variera avec l'espèce. Pour l'homme nous n'attachons d'importance qu'aux agglutinations à partir de $\frac{1}{5}$. Pour le séro-diagnostic de la tuberculose des bovidés adultes, M. Arloing ne regarde comme positif que les cas où la réaction est absolument complète à partir de la dilution à 1 p. 10.

Ce n'est donc pas seulement la constatation du pouvoir agglutinant d'un sérum et la présence d'amas dans le mélange, qui constitue un séro-diagnostic positif, puisque de tels phénomènes se produisent avec les sérums normaux, mais bien la constatation d'une agglutination à partir d'un degré de dilution où le sérum normal ne donne pas cette agglutination totale.

La question du séro-diagnostic ne repose pas seulement sur le phénomène de l'agglutination, mais bien sur le degré d'intensité de cette réaction. Et c'est parce que le degré de l'agglutination des sérums normaux n'est souvent pas très éloigné de celui des sérums tuberculeux, qu'il faut prendre une base d'appréciation sûre et toujours identique, et qui est, pour nous, la réaction macroscopique complète.

Par conséquent :

3°. Pour exprimer les résultats du séro-diagnostic nous croyons qu'il faut les diviser en trois classes :

(*a.*) *Les résultats positifs*, c'est-à-dire ceux où il y a réaction complète à un titre où le sérum normal de l'espèce envisagée ne donne pas cette réaction.

(*b.*) *Les résultats négatifs*, où il n'y a pas d'agglutination appréciable à partir du degré de dilution où le sérum normal n'agglutine plus naturellement.

(*c.*) Enfin les cas douteux, dans lesquels la réaction est seulement incomplète.

Surtout dans l'application du séro-diagnostic à la tuberculose humaine, il faut savoir parfois rester dans le doute quitte à être fixé par un ou plusieurs examens répétés ultérieurement.

On voit que dans ce chapitre de technique nous avons insisté souvent sur la nécessité de se placer toujours dans des conditions identiques, et aussi dans des conditions moyennes où les causes d'erreur soient réduites au minimum. C'est pour le motif général que nous n'employons pas des cultures trop jeunes que nous ne sommes pas très partisans de la réaction extemporanée avec mélange à parties égales, de l'examen microscopique comme critérium exclusif, ni de toute autre condition qui contribue à accélérer au delà de certaines limites la rapidité de l'agglutination ; car on s'expose ainsi à

Digitized by Google

multiplier les causes d'erreur en précipitant ou en rendant trop délicates à observer les conditions du phénomène.

En employant des cultures telles que nous l'avons dit, dans les conditions de mélange, etc., indiquées plus haut, en ne tenant pour positive que les réactions macroscopiques complètes des premières heures, on arrivera assez facilement aux résultats heureux qu'ont obtenus après nous Mongour, Buard, Mosny, Carrière, Bendix, Romberg, etc.

3e PARTIE.

RÉSULTATS—CONCLUSIONS.

Les résultats du séro-diagnostic de la tuberculose sont actuellement appuyés non seulement sur nos travaux personnels, mais sur ceux des nombreux auteurs que nous avons cités. Voici le résumé des principaux résultats.

Chez l'homme tuberculeux le sérum se comporte, pour la séro-réaction, comme celui de certains animaux tuberculisés, les cobayes par exemple. Son pouvoir agglutinant s'élève rarement au-dessus de 1 pour 20 et jamais au titre élevé que nous avons constaté chez le chien par exemple. Généralement il oscille autour de 1 pour 10 et c'est pour cela qu'il faut pour le mesurer exactement prendre les précautions indiquées.

I. Pouvoir Agglutinant du Sérum Sanguin Humain.

(A.) *Dans les cas de tuberculose dite "médicale."*

Nous avons étudié le sérum de plus de 400 sujets pris au hasard, principalement dans les services du Professeur Bondet ou du service du Professeur Tessier, à l'Hôtel-Dieu de Lyon. Voici nos résultats :

(a.) *Chez les malades tuberculeux d'après la clinique :*

Réactions positives	87·9 p. 100
Réactions négatives	12·1 „

(b.) *Chez les malades non-tuberculeux d'après la clinique :*

Réactions positives	34·6 p. 100
Réactions négatives	65·4 „

(c.) *Chez les sujets sains en apparence :*

Réactions positives	26·8 p. 100
Réactions négatives	73·2 „

Si l'on compare le chiffre des réactions positives dans les trois catégories, on voit que ce chiffre baisse graduellement en passant des tuberculeux (87·9 p. 100), aux malades hospitalisés (34·6 p. 100), et enfin aux sujets valides 26·8 p. 100), ce qui correspond à ce que l'on sait pour ces deux dernières

Digitized by Google

catégories de la fréquence de la tuberculose chez les individus en apparence indemnes.

Chez les tuberculeux nous avons pris les cas les plus disparates, comme localisation (poumons, plèvre, péritoine, intestins, ganglions, etc.), et comme gravité (depuis la phtisie cavitaire, la phtisie galopante, la pneumonie gaséeuse jusqu'à la pleurésie bénigne, et les lésions discrètes des sommets). Nous avons depuis longtemps signalé que ce sont *les tuberculoses graves qui fournissent presque tous les cas de séro-réaction négative*, et *qu'au contraire les tuberculoses discrètes et curables donnent presque toujours un séro-diagnostic positif*. MM. Mongour, Buard, Rothamel en 1899, Bendix, Romberg ont absolument confirmé et développé ce dernier point (voir pour la discussion des faits, notre article 'Zeit. f. tubercul.,' Band 1, Heft I, p. 117, et 'Congrès de Paris,' 1900).

Chez les malades non-cliniquement tuberculeux, nous avons décélé par la séro-réaction une énorme proportion de tuberculoses latentes. Ceci ne doit pas nous surprendre. Les autopsies nous ont souvent permis de confirmer un séro-diagnostic qui pendant la vie était en désaccord avec la maladie apparente et le diagnostic clinique (voir les articles déjà cités). La statistique de M. Romberg, notamment, plaide absolument dans le même sens que la nôtre (voir le travail de Romberg).

Quant à la question de savoir si une séro-réaction positive ne peut pas exister sans qu'il y ait des lésions tuberculeuses apparentes, c'est un point à réserver, car il est possible qu'une imprégnation tuberculeuse antérieure, dûe à des lésions guéries, ou bien des lésions très minimes qui échappent aux recherches, ou encore un microbisme tuberculeux latent, puisse expliquer une telle hypothèse (voir cette discussion dans notre article de la 'Gazette des Hôpitaux,' 1900).

Quant aux sujets sains en apparence, la moindre fréquence de la séro-réaction positive chez eux est encore en rapport avec la fréquence des tuberculoses latentes.

(B.) *Dans les cas de Tuberculoses dites "Chirurgicales."*

L'étude de malades pris au hasard et rapportée dans la thèse de Clément* nous a donné le résultat suivant: Chez les non-tuberculeux, la réaction a toujours été négative. Chez les tuberculeux, la séro-réaction a été positive dans les proportions suivantes: 76·6 p. 100 dans les cas de lésions ostéo-articulaires, 75 p. 100 dans les cas de lésions osseuses pures, et 70·6 p. 100 dans les cas de lésions tuberculeuses diverses.

II. Pouvoir Agglutinant des Sérosités Tuberculeuses.

La méthode générale a été étendue par l'un de nous au diagnostic de la nature des épanchements pathologiques, par la recherche du pouvoir agglutinant non seulement du sérum sanguin, mais des sérosités pathologiques elles-mêmes.

* *H. Clément.*—"Le séro-diagnostic des tuberculoses chirurgicales." Thèse. Lyon, 1900.

Digitized by Google

On trouvera dans un travail récent * tous les détails d'application et les résultats obtenus par l'étude de 135 épanchements des séreuses, tuberculeuses ou non, chez l'homme ou chez l'animal.

Le pouvoir agglutinant est toujours absent dans les sérosités non-tuberculeuses ; il est presque toujours présent, et souvent élevé dans les pleurésies, péritonites tuberculeuses bénignes ; il peut être absent dans les cas de tuberculose grave (pleurésie purulente, granulie pleurale, péritonéale, méningée, etc.). La constatation du pouvoir agglutinant d'une sérosité pleurale sera donc un argument de très grande valeur du diagnostic tuberculose.

III. Des Causes qui entraînent une Modification du Pouvoir Agglutinant chez les Tuberculeux.

Nous avons vu que les tuberculeux gravement malades ont souvent un pouvoir agglutinant nul ou très faible. On connaît des faits analogues dans l'usage de la tuberculine employée comme moyen de diagnostic ; les bovidés atteints d'une tuberculose très avancée réagissent fort mal à la tuberculine.

Pour découvrir la cause de ce fait nous avons recherché chez de nombreux animaux le pouvoir agglutinant du sérum avant et après un tuberculisation par des cultures soit très virulentes soit très atténuées. Ces expériences sont consignées dans un de nos mémoires.†

Nos conclusions générales sont les suivantes. Les agglutinations dans ces expériences paraissent subordonnées à deux facteurs : 1° La virulence de l'agent tuberculisant ; 2° La susceptibilité de l'espèce animale à la tuberculose. Le développement du pouvoir agglutinant chez un individu semble demander que cet individu résiste le plus possible à l'infection tuberculeuse. Il résistera dans deux cas : lorsque son organisme offrira naturellement un milieu peu propre à l'évolution de la tuberculose, ou bien, lorsque offrant un terrain propice, il reçoit un virus affaibli.

Sans doute il est possible que d'autres conditions interviennent dans les variations du pouvoir agglutinant tuberculeux chez l'homme ou les animaux, mais nous pensons devoir placer au 1er rang celles qui précèdent. Nous n'avons pas à insister sur les conséquences de ces faits, qui expliquent la plupart des cas de séro-réactions négatives chez l'homme, et acquièrent une certaine importance au point de vue du pronostic des tuberculoses humaines.

CONCLUSIONS GÉNÉRALES.

Nous pouvons aujourd'hui, grâce à notre statistique générale, et grâce aux expériences ou observations confirmatives faites par plusieurs auteurs,

---

* Pour les détails et l'exposé des cas, voir : Paul Courmont, "L'Agglutination du bacille de Koch par les épanchements des séreuses." ('Arch. de méd. expérim.,' nov. 1900) et Th. de Feitu sur le même sujet. Lyon, 1900.

† Voir : 'Journal de physiol. et pathol. générales,' 1900, No. 1, p. 83 ; et aussi : 'Société de Biologie,' 1er décembre 1900.

Digitized by Google

répéter avec plus de confiance que jamais les conclusions générales et les applications cliniques suivantes :

1° L'emploi de cultures homogènes ou bouillon glycériné du bacille de la tuberculose humaine nous a permis de rechercher le pouvoir agglutinant des humeurs, et principalement du sérum des tuberculeux, et d'arriver ainsi au séro-diagnostic de la tuberculose.

2° Les avantages de la méthode sont ceux du séro-diagnostic en général : (1.) Innocuité absolue, puisqu'il suffit de prélever quelques gouttes de sang ou de sérosité, et qu'elle peut s'appliquer à tous les sujets fébricitants ou non, gravement ou peu malades ; (2.) Facilité et rapidité, puisqu'il n'est pas nécessaire d'avoir le sujet sous la main, et que l'expérience ne dure que quelques heures ; (3.) Sensibilité de la réaction dans les cas de lésions discrètes ou latentes et difficiles à reconnaître par les procédés habituels.

Il est vrai que les détails de technique pour entretenir les cultures liquides convenables et appliquer le séro-diagnostic sont assez minutieuses et indispensables à suivre, en raison du peu d'élévation du pouvoir agglutinant chez l'homme tuberculeux. Nous rappelons surtout la nécessité de l'emploi du sérum-étalon.

3° Pour le cas de tuberculose pulmonaire peu avancée, le pouvoir agglutinant du sérum est presque constant, mais à des degrés divers ; il varie de 1 p. 5 à 1 p. 20 et plus.

Dans les cas graves, à lésions très étendues ou très virulentes, la séro-réaction peut manquer fréquemment ou être très faible. Elle peut aussi diminuer d'intensité et disparaître à mesure que l'état s'aggrave. Les faits expérimentaux confirment ceux de la clinique. Le pouvoir agglutinant chez les tuberculeux paraît donc le plus souvent et dans certaines limites, en raison inverse de la gravité de l'infection ou de l'étendue des lésions.

4° Chez les malades atteints d'affections diverses, et chez lesquels la clinique ne révèle pas des signes certains de tuberculose, la séro-réaction permet de déceler un grand nombre de tuberculoses latentes. L'autopsie ou l'évolution ultérieure de la maladie viennent fréquemment apporter la confirmation du séro-diagnostic.

5° De même, chez les sujets sains en apparence, la réaction agglutinante, absente le plus souvent, est positive dans un certain nombre de cas dont les chiffres correspondent à ce que l'on sait de la fréquence de la tuberculose latente.

6° Par conséquent, en pratique : une séro-réaction positive, chez un sujet suspect, sera un signe de grande valeur pour établir l'existence d'une tuberculose viscérale ; une séro-réaction négative n'aura qu'une valeur moindre comme signe négatif puisque l'agglutinant fait défaut chez un certain nombre de tuberculeux.

Mais l'absence de séro-réaction se rencontre surtout chez les tuberculeux avancés, où l'on n'a plus besoin de séro-diagnostic ; elle pourra dans certains cas confirmer un pronostic défavorable. Au contraire, chez un sujet soupçonné de tuberculose, mais sans signe clinique évident et sans symptôme de gravité, l'absence de pouvoir agglutinant paraît avoir une grande valeur pour contribuer à éliminer cette affection.

Digitized by Google

Il va sans dire que l'agglutination par le sérum sanguin ne fait pas le diagnostic de localisation de l'infection tuberculeuse; pour ce dernier point, c'est à la clinique d'utiliser les données du laboratoire.

7° En résumé, la séro-réaction nous paraît constituer un procédé rapide, inoffensif pour le malade, et d'une grande valeur pour le diagnostic précoce de la tuberculose, surtout chez les sujets suspects de lésions pulmonaires au début.

TECHNIQUE AND RESULTS OF THE SERUM-DIAGNOSIS IN TUBERCULOSIS.*

By MM. les Drs. S. ARLOING and P. COURMONT, *Lyons, France.*

The employment of homogeneous cultures of the bacilli of human tuberculosis in glycerinated broth offers a means of investigating the agglutinising power of secretions, and especially of the serum in tuberculous animals and men. In this sense the serum-diagnosis of tuberculosis is an easy and rapid method, and one absolutely innocuous to the patient. The only real difficulty consists in obtaining cultures suitable for the purpose.

Our researches in the most varied cases of human tuberculosis show that a positive serum reaction in a suspected subject will be a most valuable sign in establishing the existence of visceral tuberculosis. A negative reaction in a suspected case will help in excluding a diagnosis of tubercle. The serum-diagnosis of tubercle is therefore most valuable, especially in the preliminary diagnosis in cases suspected of early pulmonary lesions.

Many authors have verified and confirmed these facts—Ferré, Mongour, Buard, at Bordeaux; Mosny, in Paris; Bendix, Romberg, in Germany, etc. But others have not succeeded in preserving the homogeneous cultures, or, at least, have not achieved satisfactory results by their use.

This is doubtless owing to the variations in the properties of the cultures in the hands of different experimenters, and to the difficulty of estimating, in the cultures, the conditions favourable to agglutination. To obviate these difficulties we have for some time past employed cultures the growth of which has been arrested by the use of formalin at the time when they present the most favourable conditions. Large quantities of cultures ready for use may thus be prepared, and will retain their properties for a long period.

It is easy to send such cultures, specially adapted for serum reactions, long distances, and thus make a large number of reactions with an agglutinable material of an identically similar kind. By using these formalin cultures, whose properties are standardised once for all, we have obtained still more exact results, completely confirming the conclusions set forth above.

---

* *Vide:* "Congrès pour l'étude de la Tuberculose," Berlin, 1899; 'Zeitschrift f. Tuberculose u. Heilstättenwesen,' 1900, B. 2, and 'Gazette des Hôpitaux,' décembre 1900.

Digitized by Google

SERUM DIAGNOSIS OF TUBERCULOSIS IN SURGICAL CASES.

By MM. les Drs. S. ARLOING and P. COURMONT, *Lyons, France.*

Serum diagnosis, by the use of homogeneous liquid cultures, may be of great assistance in the diagnosis of the nature of surgical lesions.

The following are the statistics of the results of sixty-six observations :—

	Reactions positive.	Reactions negative.
Non-tuberculous lesions : 11 cases.	0	11
Lesions clinically tuberculous : 55 cases.	41, or 74·54 %	14, or 25·46 %

These sixty-six patients all belonged to the surgical division. The tuberculous subjects showed most diverse localisations: osteo-articular, osseous, glandular and cutaneous.

It has seemed to us that the serum reaction was more constant and frequent in cases which ran a favourable course, and that the graver cases gave a larger number of negative reactions. So that from a practical point of view a positive serum reaction is a sign of great value in favour of the existence of surgical tuberculosis.

LE SÉRO-DIAGNOSTIC DES TUBERCULOSES DITES "CHIRURGICALES."

Par MM. les Drs. S. ARLOING et PAUL COURMONT.

Le séro-diagnostic par l'emploi des cultures liquides homogènes peut rendre de grands services pour le diagnostic de la nature des lésions dites chirurgicales.

Voici les résultats d'une statistique portant sur 66 observations :—

	Réactions positives.	Réactions négatives.
Lésions non-tuberculeuses. 11 cas.	0	11
Lésions cliniquement tuberculeuses. 55 cas.	41 soit 74·54 %	14 25·46 %

Ces 66 malades appartenaient à des services de chirurgie. Les tuberculeux offraient les localisations les plus diverses : *ostéo-articulaires, osseuses, ganglionnaires et cutanées.*

Il nous a semblé que la séro-réaction était plus constante et à un taux

Digitized by Google

plus élevé dans les cas à évolution favorable, et que les cas les plus graves donnaient un plus grand nombre de séro-réactions négatives.

De sorte qu'au point de vue pratique une séro-réaction positive est un signe d'une grande valeur en faveur de l'existence d'une tuberculose chirurgicale.*

RECHERCHES SUR L'AGGLUTINATION DU BACILLE DE KOCH ET LE CYTO-DIAGNOSTIC DANS VINGT-QUATRE CAS D'ÉPANCHEMENTS SÉRO-FIBRINEUX DE LA PLÈVRE.

PAR MM. LES DRS. WIDAL ET RAVANT.

NOUS avons eu l'occasion de rechercher l'agglutination du bacille de Koch par des liquides pleuraux de natures diverses, au moyen du bacille mobile de MM. Arloing et Courmont; la technique que nous avons suivie est celle qu'ont indiquée ces auteurs dans différents travaux.†

Un point de technique nous semble capital: c'est de ne point pratiquer de séro-réaction sans avoir essayé la culture avec un sérum témoin connu; cette précaution est absolument indispensable, car de jour au jour le pouvoir agglutinant des cultures varie considérablement. Celles qui nou sont paru donner les réactions les plus nettes sont des cultures peu épaisses et datant de 5 à 6 jours.

Dans ces conditions nous avons recherché dans 24 liquides pleuraux séro-fibrineux la séro-réaction; les résultats que nous avons obtenus sont les suivants :—

Sur 11 *pleurésies idiopathiques ou à frigore, et dont la nature est tuberculeuse, la séro-réaction a été positive* 9 *fois*, douteuse 1 fois, et négative 1 fois.

Sur 2 pleurésies de phtisiques : deux résultats négatifs.

Sur 11 *pleurésies diverses dont aucun n'était tuberculeuse:* 11 *résultats négatifs.*

Ces faits nous montrent que cette séro-reaction, faite avec les soins et les précautions nécessaires, peut fournir des renseignements précis sur la nature d'un épanchement pleural.

Le cyto-diagnostic de ces liquides pratiqué selon la méthode que nous avons déja indiquée, nous a montré que les 11 pleurésies idiopathiques avaient une formule lymphocytique et qu'aucune d'elles ne renfermait de cellules endotheliales; l'absence de ces derniers éléments et la présence presque exclusive de lymphocytes dans un épanchement séro-fibrineux est la formule de la pleurésie idiopathique, dont la nature est tuberculeuse et dont la

* On trouvera les observations *in extenso* de cette statisque dans la thèse de notre élève *Clément: Le séro-diagnostic des tuberculoses dites chirurgicales*, Lyon, 1900.

† Voir: *P. Courmont.*—"Le Séro-diagnostic des Épanchements Tuberculeux." 'Archiv. de méd. expérim.,' No. 6, 1900.

Digitized by Google

caractéristique anatomique est la présence d'une néo-membrane qui par sa formation empêche ainsi la desquamation endotheliale.

Au contraire, les 11 autres pleurésies non-tuberculeuses, et de natures diverses, renfermaient des cellules endotheliales soudées, des polynucléaires, et quelques lymphocytes dans des proportions variables : leur formule histologique était totalement différente de celles de la variété précédente.

THE AGGLUTINATING REACTION IN CASES OF PULMONARY TUBERCULOSIS.

By P. HORTON-SMITH, M.D., F.R.C.P., and H. W. ARMIT, M.R.C.S., L.R.C.P.

THE work of Professors Arloing and Courmont upon this subject is well known.* It is work that merits our most earnest attention, for of its importance there can be no question. Everyone who has had much to do with pulmonary tuberculosis knows well that not infrequently cases occur in which the diagnosis is in doubt, and cannot be decided by the discovery of the specific bacilli in the sputum, owing to the fact that no expectoration is brought up. If, then, in a case of this kind the serum could be shown to possess demonstrable and specific agglutinating power, such a test would become of the highest practical value.

Through the kindness of Professor Courmont, who was good enough to present to us a subculture of his homogeneous tubercle bacillus, and to whom we desire to express our great indebtedness, we have been enabled recently to work at this subject.

It is with the greatest regret, therefore, that we now feel ourselves obliged to state that we have been unable so far to confirm the results obtained by Professors Arloing and Courmont.

Speaking generally, these observers have found that all cases of pulmonary tuberculosis, except those that are quite moribund, agglutinate well, and for the most part in dilutions of 1·10 or higher.

Thus (1) of 26 advanced cases quoted by them in a paper read at the Paris Congress of Tuberculosis in 1898, 69 per cent. agglutinated in a dilution of 1·10 or 1·20, and

(2) of 22 mild cases, 82 per cent. agglutinated in similar dilutions;

making an average roughly of about 75 per cent.

What now are the results which Mr. Armit and myself have obtained?

We have examined 29 *cases of Pulmonary Tuberculosis*, all with physical signs, but all able to be up and about; that is to say, none were moribund, and therefore unlikely to agglutinate, according to the view enunciated by our French *confrères*.

* See especially *Le Séro-Diagnostic de la Tuberculose*. 'Gazette des Hôpitaux,' Dec. 1, 1900. References to earlier publications on the subject will be found in this Paper.

Digitized by Google

Of these twenty-nine cases—

3 gave no reaction in a 1·5 dilution . .	10·34 per cent.		
*5 gave a trace „ „ . .	17·24 „	89·64	
*11 gave a partial „ „ . .	37·93 „		
*7 gave a complete „ „ . .	24·13 „		
3 „ „ 1·10 dilution .	10·34 „		10·34

Hence, *since a reaction, even if complete, in a* 1·5 *dilution is not specific, as Courmont and Arloing allow*, we have the result that roughly 90 per cent. of our cases gave no practical result, while only 10 per cent. gave what Arloing and Courmont would call a positive (specific) reaction. It may be added too, that, contrary to the results obtained by the French observers, our three positive cases were patients with sufficiently advanced disease (one having also tubercular laryngitis), while two very early cases without physical signs, which Professors Arloing and Courmont find in general give strong agglutinating reactions, in our hands yielded negative results. (These two cases were not included in the above series.)

Consideration of Results.—These results are unfortunately very different to those of Professors Arloing and Courmont. It may be asked, therefore, whether we have fallen into any error in our technique. We believe that this is not so. For, in the first place, *the cases referred to above* were all patients who undoubtedly had phthisis, as found by physical examination, *and by the discovery of tubercle bacilli in their sputum in every case.* Further, that our culture was in good working order was proved by the fact that *on each occasion before being used* it was tested against serum from an inoculated goat kindly placed at our disposal by Professor Courmont, and was found to agglutinate even in a 1·50 dilution. The technique, moreover, used by us was similar to that advocated by Professor Courmont, dilutions of 1·5, 1·10, and 1·20 being made, and examined after one, two, three, four, and twenty-four hours. On one point only was a modification made, namely, that on *two* occasions we used a culture eight days old, *killed by the addition of formalin* (1·150). As, however, this culture agglutinated exactly as did the living culture when treated with the standard goat's serum referred to above, we believe that no error was thereby introduced. Although, then, our results are so different from those obtained by the introducers of the method, and therefore invite criticism, we believe that they cannot be explained as the result of any error in procedure, but are on the contrary substantially accurate. It should be added, too, that they are in close agreement with those obtained by *Beck* and *Rabinowitsch*.† These observers found that of

17 commencing cases of phthisis, only 2 reacted in dilutions of 1·10, or higher	11·7 per cent.
while of 16 advanced cases, 2 only reacted in similar dilutions	12·5 per cent.

* None of these cases showed any sign of agglutination in dilutions of 1·10 and 1·20.

† *Über den Werth der Courmont'schen Serum-Reaction für die Früh-diagnose der Tuberkulose.* 'Deutsch. med. Wochenschrift,' 1900, No. 25, s. 400.

Digitized by Google

Roughly, therefore, of all cases of pulmonary tuberculosis examined by them, 12 per cent. only gave a reaction in a dilution of 1·10 or higher, a result which very closely approximates with the 10·34 per cent. obtained by ourselves.

Conclusion.—According therefore to the results which we have so far obtained, and which closely agree with those of the Berlin investigators, it would seem that *we may only expect a reaction in dilutions of* 1·10 *or higher* in some 10 *per cent. of all cases of pulmonary tuberculosis*, as opposed to the 75 per cent. observed by Professors Arloing and Courmont.

Whether even then such agglutination (obtained in dilutions of 1·10 or higher) is to be regarded as specific, and can therefore be of any use in diagnosis even in this small percentage of cases, is a matter to which we propose to return at a later date. On this occasion we desire to lay stress upon the fact that *an agglutinating reaction in dilutions higher than* 1·5 *seems to us to occur in only a very small proportion of cases of pulmonary tuberculosis, and that its possible value, therefore, as a means of diagnosis becomes but slight.*

By Frau Dr. Med. LYDIA RABINOWITSCH (*Berlin*).

Professor Beck and myself obtained our cultures directly from Courmont and Arloing, as well as from Professor Ehrlich, Professor C. Fraenkel and Dr. Bendia. The experiments made by us with these homogeneous cultures and human blood-serum, as well as with blood-serum from cattle, proved that it is *not* possible to diagnose tuberculosis by the positive or negative result of the agglutination test. The agglutination proved often positive where there was no trace of tuberculosis, and on the other hand we have often had negative results in cases of incipient tuberculosis. I shall not repeat here our experiments upon human blood-serum, as well as upon blood-serum from cattle, published by us during the last two years in the *Deutsche medicinische Wochenschrift* and in the *Zeitschrift für Hygiene und Infections-Krankheiten.* I shall only add that it was a great pleasure for me to hear the paper of Dr. Courmont and to ascertain myself once more that there were no mistakes made by us referable to the technique of these experiments.

DE LA SÉRO-RÉACTION COMME MOYEN DE DIAGNOSTIC DE LA TUBERCULOSE. DES DIVERS MOYENS DE LA PRATIQUER.

Par M. le Dr. G. BUARD.

Préparateur en médecine expérimentale à la Faculté de Médecine de Bordeaux.

En 1898 M. le professeur Arloing (de Lyons) publiait les premiers résultats de ses recherches sur l'agglutination du bacille tuberculeux par le sérum sanguin ou les sérosités de tuberculeux. Afin d'observer ce phénomène, il

Digitized by Google

montra tout d'abord qu'il était nécessaire d'avoir des cultures particulières, cultures homogènes, dans lesquelles le bacille de Koch se trouve à l'état d'éléments isolés et mobiles.

Dès 1899, après avoir essayé cette agglutination avec des cultures adressées par M. Arloing, reprenant cette étude, nous avons pu obtenir sur pommes de terre glycérinées des colonies humides, qui après plusieurs passages nous permettent d'obtenir des cultures homogènes en bouillon glycériné. Ces tubes de culture nous les avons préparés très simplement, en prenant des pommes de terre de bonne qualité qui, après avoir été bien lavées et pelées, ont été coupées de façon à obtenir des morceaux rectangulaires, présentant une face plane bien disposée pour l'ensemencement. Ces morceaux nous les faisions pénétrer dans des tubes rétrécis à leur quart inférieur et nous y versions de l'eau glycérinée à 6 pour cent de manière à ce que la surface libre du liquide vienne affleurer au bord inférieur des pommes de terre. Elles séjournaient alors à l'autoclave à 120° pendant *trente minutes*. Suivant toutes les indications données par M. Arloing, nous avons pu obtenir, avec ces cultures, en bouillon glycériné, une culture homogène du bacille tuberculeux.

Avec de grosses carottes à jus, préparées comme nous venons de l'indiquer pour les pommes de terre, nous avons pu obtenir après un séjour de *quarante-cinq minutes* à l'autoclave à 220° un milieu très propice à la culture du bacille de Koch. À l'étuve à 38° il a poussé sur ce milieu de belle colonies en une dizaine de jours et après plusieurs passages successifs nous avons pu obtenir des colonies très humides qui nous ont permis de passer du milieu solide au milieu liquide. Lorsqu'on reporte la culture en bouillon sur ces carottes, on obtient très rapidement de fort belles colonies blanches, très humides, qui croissent longtemps et abondamment, et redonnent très facilement des cultures homogènes.

Dernièrement, nous avons pu voir pousser convenablement le bacille tuberculeux sur un autre milieu végétal, plus sucré que la carotte, sur la betterave. Nous avons préparé ce végétal comme les carottes, dans des tubes dits à pommes de terre, en présence d'eau glycérinée à 6 pour cent, puis nous les avons portés à l'autoclave à 120° pendant *quarante-cinq minutes*. Ce milieu est d'une couleur violet foncé où la culture blanche du bacille de Koch est blanche nettement. Il semble y pousser facilement en voile, et s'émulsionne bien dans le bouillon glycériné.

Toutes ces cultures nous ont permis de faire des remarques qui ont leur importance au point de vue de la morphologie du bacille. Les formes courtes sont de beaucoup les plus nombreuses, surtout dans les cultures jeunes. Ces bacilles sont plus grêles, plus allongés, et même effilés. Lorsqu'on les colore par la méthode de Ziehl, presque tous présentent dans le corps cellulaire des points surcolorés, et quelquefois en si grand nombre et si rapprochés que l'on dirait de véritables réunions de microcoques en chaînette. Un examen attentif montre que ce sont des éléments d'un même bacille. Les cultures en bouillon de leur côté se développent d'autant plus rapidement que les réensemencements sont plus fréquents ; d'autre part, plus le développement est rapide, plus le bacille est mobile, et moins il a de tendance à former

Digitized by Google

des pseudo-agglutinats. Les cultures jeunes et pauci-bacillaires sont les plus propices à la pratique de la séro-réaction.

Au Congrès de Montpellier (1898) M. le professeur Arloing publiait les premiers résultats qui lui ont permis de regarder la séro-réaction comme un moyen de diagnostic de la tuberculose. MM. Arloing et Paul Courmont, au Congrès de la Tuberculose de Paris (1898) et à l'Académie des Sciences (1898) produisent des nouveaux résultats. Ils montrent que la réaction est d'autant plus marquée que les lésions sont moins développées, tandis que le pouvoir agglutinatif peut manquer chez certains phtisiques arrivés à la dernière période.

M. Monyon et moi, nous avons produit dans des notes successives à la Société de Biologie (octobre 1898, juin et juillet 1899) les résultats que nous obtenions par cette méthode. Nous devons reconnaître que plusieurs fois cette séro-réaction a dévoilé des tuberculoses qui avaient passé jusqu'à ce jour inaperçues. Cette étude nous a permis en outre d'observer que la séro-réaction est d'autant plus positive que le sujet atteint est plus résistant ou plus éloigné de la cachexie (Société de Biologie, 24 juin 1899). Le docteur Rothamel, dans sa thèse inaugurale (Bordeaux, novembre 1899), montre bien que l'agglutination du bacille de Koch, mobile par le sérum sanguin du tuberculeux, est d'autant moins intense que le malade est plus près de la cachexie.

Ces différents, travaux confirmant ceux de MM. Arloing et P. Courmont, permettent d'énoncer l'hypothèse que cette agglutination semble correspondre à un phénomène de défense de l'organisme.

Poursuivant cette étude de la séro-réaction tuberculeuse, nous avons recherché chez l'enfant si le sérum sanguin pouvait agglutiner le bacille mobile (Bordeaux, mai 1900). Nos résultats sont venus montrer à nouveau combien est net ce phénomène de la séro-réaction.

N'oubliant pas ce fait que le sérum sanguin d'individu normal peut présenter un certain pouvoir agglutinatif, vis-à-vis du bacille tuberculeux mobile, nous avons employé dans toutes nos recherches des procédés qui les rendent comparables entre elles.

Pour cela nous avons toujours mélangé à des volumes constants de culture des doses constantes de sérum et de plus nous avons comparé le temps dans lequel l'agglutination s'est produite au temps maximum dans lequel des mélanges identiques se sont agglutinés avec des sérums normaux. Ces expériences primordiales ont été la base de la méthode, et ainsi tous les phénomènes d'agglutination nette se produisant dans un temps moindre peuvent être considéré comme positifs. Il est donc absolument nécessaire que chaque expérimentateur connaisse pour ses cultures ce temps maximum.

En outre comme nous avions l'habitude de le faire pour la fièvre typhoïde, nous avons vu que cette séro-réaction par le sérum sanguin en préparation extemporanée goutte par goutte donne de bons résultats, mais il est bien nécessaire de calculer également au préalable le temps d'agglutination de sa culture avec le sérum normal mélangé dans les mêmes conditions.

Le sang recueilli sur papier stérilisé et desséché conserve également son

Digitized by Google

pouvoir agglutinatif vis-à-vis du bacille tuberculeux ; il agglutine, mais plus faiblement que le sérum, c'est pourquoi nous mélangeons deux gouttes de sang réhumidifié avec une goutte de culture. Comme pour le sérum sanguin, il faut évidemment que l'observateur connaisse le temps nécessaire à l'agglutination de sa culture avec le sang de l'individu normal recueilli de la même façon. Nous avons montré plusieurs fois combien on pouvait attendre de ce procédé, et le docteur A. Moumayon a dans sa thèse inaugurale (Bordeaux, mars 1901) montré combien ce procédé était sûr, et combien on pouvait compter sur les résultats obtenus. En réunissant 64 observations il a pu voir qu'aucune expérience ne s'est trouvée en contradiction avec le diagnostic clinique.

Ces deux opérations (mélange de culture et de sérum goutte par goutte, pratique du séro-diagnostic avec le sang desséché) faites d'après les conseils de M. le professeur Ferré, ont été fréquemment répétées, dans le but de faciliter le diagnostic de la tuberculose à tout médecin de notre ville ou de la région pendant ces deux dernières années. Nous avons pu remarquer que tous les liquides de l'organisme permettent de pratiquer cette séro-réaction.

Cette méthode de diagnostic de la tuberculose que nous pratiquons depuis 1898 dans le laboratoire de M. le professeur Ferré, nous a permis de constater toute la valeur du procédé : sur 50 cas publiés dès 1899, deux fois seulement la méthode s'est trouvée en défaut ; dans un cas, le séro-diagnostic a été négatif, et cependant il s'agissait d'une pleurésie expérimentalement reconnue tuberculeuse ; dans le second cas, il fut positif chez un addisonnien sous tuberculose à l'autopsie. Le docteur Rothamel montre que chez 20 malades choisis dans des conditions particulières, et dont nous avons déjà parlé la séro-réaction s'est trouvée six fois négative, extrêmement faible ou douteuse chez 13 cachectiques, positive chez les 7 autres qui n'étaient pas cachectisés. Dans notre thèse inaugurale sur 25 cas de tuberculose infantile, tant médicale que chirurgicale, nous n'avons pas trouvé la méthode en defaut. Sur 64 examens consignés dans la thèse du Dr. A. Moumayon il a pu montrer que les résultats concordaient toujours avec la clinique (dans ces cas, il n'y avait pas de cachectiques). Donc sur 159 expériences, en laissant de côté les cas de cachexie tuberculeuse où le séro-diagnostic est suspect, la séro-réaction a donné des résultats concordant avec la clinique ou avec l'anatomie pathologique sauf dans deux cas.

Tous ces faits nous permettent donc de considérer dans l'état actuel de nos connaissances la séro-réaction tuberculeuse comme un des meilleurs moyens que nous ayons de dévoiler la tuberculose dès le début ; il donne de résultats très précieuses, et c'est même le seul à employer quand on ne peut déceler par les procédés habituels la présence du bacille de Koch.

Digitized by Google

LA PROPHYLAXIE DE LA TUBERCULOSE PULMONAIRE PAR LA CONNAISSANCE DE SON TERRAIN.

PAR MM. LES DRS. ALBERT ROBIN ET MAURICE BINET.

I.

DANS les études faites depuis vingt ans sur la tuberculose on a singulièrement laissé de côté la question du terrain de la maladie. La connaissance de la contagion et de son agent a orienté la prophylaxie et la thérapeutique de la phtisie dans la poursuite du microbe, et l'on a relégué sur un plan lointain la recherche des conditions prédisposantes qui rendent efficace l'intervention de ce microbe. Car, même parmi les individus placés dans les milieux les plus favorables à la contagion, le plus grand nombre semble échapper à celle-ci et quelle est la personne qui, dans les grands centres de population, n'a pas été plus ou moins exposée à faire pénétrer dans ses poumons de l'air chargé de poussières bacillifères?

Pour qu'on devienne tuberculeux, il faut donc quelque chose de plus que l'agent de la contagion, auquel nous sommes tous plus ou moins exposés. Il faut un terrain favorable à la vie et à la multiplication du bacille de la tuberculose. Si l'on parvient à déterminer ce terrain, on réalisera un immense progrès dans la prophylaxie et dans le traitement de la maladie, parce que l'on pourra entourer de plus de précautions contre la contagion ceux qui présenteront les stigmates de ce terrain, et parce que l'on conçoit l'espérance de trouver des agents capables de rendre le terrain réfractaire.

II.

Or, nous avons démontré précédemment que par l'analyse des échanges respiratoires on pouvait non seulement faire le diagnostic du terrain prétuberculeux, mais encore saisir le pourquoi de ce terrain, c'est-à-dire l'une des raisons de l'aptitude à la tuberculose.

1°. Sur 92 pour cent des phtisiques, la capacité respiratoire est diminuée, soit absolument, soit par rapport au centimètre de taille du sujet; la ventilation pulmonaire croît de plus de 80 pour cent; l'acide carbonique de 64 pour cent; l'oxygène total consommé de 70 pour cent; l'oxygène absorbé par les tissus de 94 pour cent.* Qu'il s'agisse de phtisie aiguë ou chronique, que le malade soit examiné au début ou à la fin de la maladie, *les échanges respiratoires* sont toujours augmentés dans de variables proportions.

2°. Nous avons recherché si cette exagération des échanges respiratoires n'existait pas aussi dans d'autres maladies. Les recherches poursuivies dans quarante états morbides ont montré que si un certain nombre de ceux-ci présentaient aussi des échanges respiratoires exagérés en bloc, cependant dans chacun d'eux le chimisme respiratoire différait par quelque trait de celui

* Ces mesures sont données par kilog. de poids et par minute pour l'homme. Chez la femme, les augmentations sont encore plus considérables.

Digitized by Google

de la phtisie, ce qui permet de l'utiliser pour le diagnostic et même pour le *diagnostic précoce* de la phtisie, puisque ses caractères sont significatifs dès le début de celle-ci.

3°. Pour expliquer cet accroissement des échanges gazeux trois hypothèses se présentaient. On pouvait songer, en effet, à une *réaction de défense organique* à l'encontre du bacille, à une *conséquence de l'attaque bacillaire*, ou enfin à une manifestation du *terrain de la tuberculose.* Aujourd'hui, cette dernière hypothèse est passée à l'état de fait puisque nous avons découvert que nombre des descendants de tuberculeux avaient déjà des échanges respiratoires exagérés, que dans les états antagonistes de la phtisie, comme l'arthritisme, ils étaient au contraire diminués, ainsi d'ailleurs que dans la scrofule, ce terrain où ne germent que des tuberculoses locales.

L'exagération des échanges respiratoires est donc l'une des conditions du terrain de la phtisie. *La prédisposition, qu'elle soit héréditaire ou acquise, reconnaît, au moins comme l'une de ses causes, l'aptitude de l'organisme à consommer trop d'oxygène, à en fixer trop dans les tissus, et à produire trop d'acide carbonique, en un mot, à se consumer exagérément*, ce qui correspond bien à l'idée hippocratique persistant encore dans la tradition populaire.

III.

Pour démontrer définitivement cette proportion, examinons d'abord ce qui se passe chez les *descendants de phtisiques.* Nous avons analysé les échanges respiratoires de 28 descendants de tuberculeux paraissant jouir d'une bonne santé, indemnes de tout soupçon de tuberculose actuelle. Sept d'entre eux étaient âgés de 8 à 14 ans; les 21 autres avaient de 17 à 50 ans.

Dans les 7 premiers cas, les échanges ne sont pas exagérés. Nous nous demandons s'il n'y aurait pas là quelque relation avec ce fait que l'aptitude à la phtisie se manifeste moins avant la quinzième année, d'après la statistique de Leudet et de Hanot, et aussi d'après l'observation courante de la majorité des médecins. Remarquons aussi que chez les sujets au-dessous de 15 ans les échanges respiratoires sont très actifs; ce n'est guère qu'à partir de la 16e année qu'ils prennent leur type définitif. Enfin, les résultats de l'analyse chez les sujets de cet âge sont souvent sujets à caution, à cause de la difficulté que l'on éprouve à obtenir chez eux une respiration régulière pendant l'expérience. Nous réservons ces 7 observations jusqu'à ce que nous ayons eu l'occasion de pratiquer un plus grand nombre d'analyses.

Dans les 21 cas du second groupe (héréditaires de 16 à 50 ans), les échanges respiratoires sont augmentés 13 fois, normaux 7 fois, abaissés 1 fois, ce qui correspond au pourcentage suivant.

Les échanges respiratoires chez 21 héréditaires bien portants.

Echanges exagérés	13 fois	soit 61·9 pour cent.	
„ normaux	7 „	„ 33·3	„
„ abaissés	1 „	„ 4·8	„

Il résulte de ce tableau que sur 10 descendants de phtisiques, 6 présentent un terrain favorable à la contagion.

Digitized by Google

IV.

Nous ne saurions dire dans combien de cas *cette prédisposition se réalise*, parce que nos sujets n'ont pas encore été suivis pendant assez longtemps. Mais sur nos 13 cas à chimisme exagéré, deux sont devenus phtisiques. Le premier, examiné en avril 1896 et absolument sain à cette époque, a été emporté en juillet 1898 par une phtisie à marche rapide ayant débuté en janvier de la même année. Un frère âgé de 20 ans, examiné à la même époque, a des échanges normaux. Nous l'avons revu en juin 1901, bien portant et vigoureux. Le second présente, en octobre 1897, des échanges exagérés; en décembre 1899, l'exagération s'accentue, et en novembre 1900, on constate les premiers signes de la phtisie. Une sœur plus jeune, examinée le même jour, a des échanges physiologiques. Elle est aujourd'hui mariée et très bien portante.

Quand bien même l'on contesterait la valeur du chimisme respiratoire comme manifestation du terrain tuberculeux, on ne peut nier, tout au moins, son *importance révélatrice de la prétuberculose pulmonaire.*

Par contre, voilà une fille robuste dont le père est phtisique, qui est la seule survivante de cinq enfants morts de tuberculose méningée ; ses échanges sont normaux. Un individu de 50 ans, fils de deux phtisiques, dont les deux sœurs et le frère sont morts phtisiques, a des échanges respiratoires diminués. Or, il n'a jamais été malade, et il est un type de vigueur et de santé.

V.

Dans les 21 cas du deuxième groupe, nous n'avons réuni que des sujets ayant toutes les apparences de la santé. Voici maintenant 6 cas où des *descendants de phtisiques étaient atteints de maladies déterminées.*

1°. Un jeune homme de 21 ans, sans hérédité directe, mais avec une grand'mère morte de phtisie, vient d'avoir la *grippe.* Il est très amaigri, continue de tousser, a des sueurs nocturnes; on soupçonne un début de phtisie. L'analyse des échanges respiratoires donne des chiffres à peu près normaux :

Acide carbonique produit . . .	4″·751
Oxygène consommé total . . .	5″·938
„ absorbé par les tissus . .	1″·187

Revu après deux années, il est en parfaite santé.

2°. Une jeune fille de 19 ans a perdu sa mère de phtisie quatre ans auparavant. Elle est atteinte d'une *grippe traînante* qui se complique de foyers disséminés et successifs de *broncho-pneumonie* avec mauvais état général et grandes oscillations thermiques. On pense à une tuberculose pulmonaire aiguë. L'abaissement des échanges fait écarter ce diagnostic.

Acide carbonique	3″·800
Oxygène consommé total . . .	4″·307
„ absorbé par les tissus . .	0″·507

Digitized by Google

La malade est guérie depuis dix-huit mois et se porte bien.

3°. Une jeune fille *chlorotique* de 16 ans est fille d'un père mort de phtisie ; elle a eu 3 frères morts de méningite tuberculeuse. Une autre *chlorotique* de 17 ans est fille d'une mère morte phtisique ; sa sœur est morte à 20 ans de tuberculose aiguë. Toutes deux ont des échanges exagérés :

	1er cas.	2e cas.
Acide carbonique produit . .	7″·004	7″·676
Oxygène consommé total . .	8″·140	10″·232
„ absorbé par les tissus .	1″·136	2″·558

La moyenne des échanges respiratoires chez 11 chlorotiques donne les chiffres suivants :

Acide carbonique produit . .	6″·148
Oxygène consommé total . .	7″·925
„ absorbé par les tissus .	1″·777

Les échanges de ces deux jeunes filles sont donc exagérés par rapport à ceux de la chlorose chez les non-héréditaires.

4°. Une femme de 50 ans, atteinte de *cancer de l'estomac* avec phlegmon consécutif de l'ombilic, fille d'une mère phtisique, a des échanges exagérés :

Acide carbonique produit . .	8″·305
Oxygène consommé total . .	11″·525
„ absorbé par les tissus .	3″·230

En général, les échanges sont ralentis chez les cancéreux, ainsi qu'en témoignent les chiffres suivants, moyennes de 4 cas :

Acide carbonique produit . .	3″·291
Oxygène consommé total . .	4″·442
„ absorbé par les tissus .	1″·151

5°. Un *saturnin* de 35 ans, d'hérédité mal établie, mais ayant perdu deux frères et une sœur de phtisie, a des échanges normaux :

Acide carbonique produit . .	4″·359
Oxygène consommé total . .	5″·605
„ absorbé par les tissus .	1″·246

On ne peut pas plus s'attendre à trouver des échanges élevés chez tous les héréditaires malades que chez tous les héréditaires bien portants. Mais il est évident que 3 malades sur 6 conservaient—alors que leur maladie ne comporte pas un chimisme aussi élevé—des *échanges indicateurs de terrain tuberculeux qu'ils tenaient de leur hérédité*.

Digitized by Google

En réunissant ces cas aux 21 précédents, on obtient l'ensemble ci-dessous : Les échanges respiratoires chez 27 héréditaires sains ou malades :

Échanges exagérés	16 fois	soit 59·25 pour cent.	
„ normaux	9 „	„ 33·33 „	„
„ diminués	2 „	„ 7·42 „	„

Cette proportion concorde sensiblement avec celle qui ressort de la statistique précédente et confirme sa valeur.

VI.

Les descendants de tuberculeux ne sont pas seuls à présenter cette suractivité des échanges respiratoires qui caractèrise au moins l'une des aptitudes à la phtisie. Cette aptitude du terrain peut aussi être acquise. On sait que les alcooliques, les surmenés, les gens mal nourris fournissent un fort contingent à la tuberculose. Si l'importance de chacune de ces conditions n'est pas la même pour tous les observateurs, aucune d'elles, toutefois, n'est mise en doute par personne. Mais, ce que l'on ne connaît pas, c'est le trouble nutritif qui caractérise ces états protopathiques de la phtisie, et l'on ne sait pas, par conséquent, si ce trouble est le même pour tous les états en question.

Or, nos recherches nous ont démontré ce fait capital, que chez *les alcooliques et les surmenés les échanges respiratoires subissaient une suractivité comparable à celle observée chez la plupart des descendants de tuberculeux.*

1°. Deux jeunes gens de 24 et 25 ans, sans tare héréditaire, jouissant d'une parfaite santé, vivant dans les meilleures conditions d'hygiène, se livrent pendant plusieurs mois à des *excès génitaux*. Leurs échanges respiratoires s'élèvent dans une forte proportion :

	1er cas (24 ans). Par kilog. minute.	2e cas (25 ans).
Acide carbonique produit	7″·067	7″·165
Oxygène total consommé	10″·460	8″·424
„ absorbé par les tissus	3″·393	1″·259

2°. Le *surmenage intellectuel* agit dans le même sens. Voici un médecin de 40 ans, très occupé déjà par sa vie professionnelle, qui se livre à des travaux intellectuels très absorbants, et voit ses échanges respiratoires s'élever sensiblement. De par ce surcroît de travail, l'acide carbonique passe de 4″·651 à 6″·843, et l'oxygène total consommé de 6″·511 à 8″·490.

3°. On sait que le *travail musculaire* active les échanges respiratoires. Il en est de même de *l'insomnie* et des *veilles*. Nous avons recueilli nombre d'observations qui confirment cette exagération. L'une d'entre elles concerne un ouvrier faïencier, âgé de 25 ans, surmené par le travail de nuit et ne parvenant pas à compenser suffisamment, par le sommeil interrompu du jour, la fatigue et l'insomnie de la nuit. Les échanges respiratoires sont considérables :

	Par kilog. minute.
Acide carbonique produit	6″·942
Oxygène consommé total	9″·029
„ absorbé par les tissus	2″·086

Digitized by Google

4°. *L'alcoolisme* agit comme le surmenage. Un garçon marchand de vins de 33 ans, buveur de vin, d'apéritifs et d'absinthe, ayant les stigmates de l'alcoolisme professionnel, quoique ne se grisant jamais, pâle, amaigri, déprimé, véritable proie pour la tuberculose, présente le chimisme suivant :

	Par kilog. minute.
Acide carbonique produit . .	7″·765
Oxygène consommé total . .	10″·358
„ absorbé par les tissus .	2″·593

5°. Une jeune femme de 28 ans, pour noyer des chagrins, boit chaque jour depuis près d'une année 5 à 6 verres *d'absinthe* sans compter le reste. Elle mange mal, souffre de l'estomac et commence à se cachectiser. Ses échanges respiratoires sont énormes :

	Par kilog. minute.
Acide carbonique produit . .	8″·359
Oxygène consommé total . .	11″·063
„ absorbé par les tissus .	2″·704

6°. *Quand l'alcoolisme et les divers modes de surmenage se combinent*, l'exagération des échanges peut atteindre des proportions tout à fait exagérées. Ainsi, un homme de 43 ans, encore vigoureux, avec quelques signes de déchéance physique, travaille dans une raffinerie, exposé à une immense chaleur, se fatiguant beaucoup et s'alcoolisant de même. Ses échanges respiratoires atteignent les hauts chiffres suivants :

	Par kilog. minute.
Acide carbonique produit . .	9″·287
Oxygène consommé total . .	11″·764
„ absorbé par les tissus .	2″·477

Ces recherches faites sur 14 sujets alcooliques ou surmenés physiquement, intellectuellement ou génitalement, aboutissent au même résultat, et nous n'avons trouvé qu'un seul cas contradictoire, chez une institutrice de 22 ans qui, quoique surmenée professionnellement, avait des échanges respiratoires à peu près normaux.

VII.

Laissons de côté toute interprétation théorique des faits précédents et ne nous attachons qu'à ces faits eux-mêmes qui sont assez nombreux pour que les hypothèses de série ou de coincidence soient écartées.

Les échanges respiratoires sont accrus chez 92 pour cent des phtisiques avérés. Ils sont également exagérés chez 60 pour cent environ des descendants de phtisiques. L'alcoolisme et les différents modes de surmenage qui sont, de par l'observation universelle, des conditions prédisposantes de la phtisie, se caractérisent aussi par une augmentation de la consommation de l'oxygène et de la production de l'acide carbonique. Tels sont les faits.

L'aptitude exagérée de l'organisme à fixer de l'oxygène et à faire de l'acide carbonique, c'est à dire à se *consumer*, constitue donc au moins l'une des caractéristiques des états protopathiques de la phtisie.

Digitized by Google

Si diverse que soit l'origine de la prédisposition, qu'il s'agisse d'hérédité, d'alcoolisme ou d'un des modes de surmenage, on retrouve cette consommation exagérée de l'oxygène et cette formation en excès de l'acide carbonique. La découverte de ces deux termes éclaire donc le mystère des états protopathiques de la phtisie que l'on désignait jusqu'ici sous le nom vague *d'états de déchéance organique.* Nous connaissons maintenant l'un des actes chimiques communs à ces états de déchéance; nous pouvons mesurer exactement son intensité et rendre à l'hérédité, par exemple, une partie de la valeur dont semblait l'avoir dépossédée la découverte du bacille de Koch.

VIII.

Parmi les *conséquences* qui résultent des faits précédents, il en est quatre dont l'importance pratique est considérable.

1°. La presque constance (92 pour cent) de l'exagération des échanges à toutes les périodes de la phtisie confirmée aidera à en réaliser le *diagnostic précoce*, et dans les cas où l'on hésite entre la phtisie et une autre affection, le chimisme respiratoire résoudra souvent la difficulté. Dans un précédent mémoire, nous en avons cité des exemples, entre autres à propos du diagnostic entre la pleurésie simple et la pleurésie tuberculeuse. Depuis, nous avons encore recueilli plusieurs observations dont l'analyse fera l'objet d'un travail spécial.

2°. On peut aussi, de par les échanges respiratoires, *diagnostiquer la prédisposition à la tuberculose.* Parmi les descendants d'une souche tuberculeuse, on reconnaîtra ceux qui sont aptes à l'infection. Depuis 7 années que nous poursuivons nos recherches, nous avons vu se réaliser deux fois notre diagnostic de la prédisposition héréditaire.

3°. Le terme vague de "déchéance organique héréditaire ou acquise," dont on s'est servi jusqu'à présent pour définir le terrain de la phtisie, ne comporte aucune indication prophylactique et thérapeutique. On dit aux alcooliques de ne plus boire, aux surmenés de se reposer, et aux descendants de tuberculeux de se tonifier; mais le propre des indications dites toniques est justement de stimuler les échanges organiques qui sont déjà, chez ces héréditaires, en état de suractivité. Au contraire, l'analyse des échanges respiratoires révèle une condition commune aux états de prédisposition et dont la connaissance comporte une nette indication thérapeutique qui est de restreindre le pouvoir de l'organisme à fixer trop d'oxygène et à faire trop d'acide carbonique. Il ne n'agit donc plus que de rechercher quels sont les *médications* et les *médicaments* capables de réaliser cette indication. Nous avons étudié déjà, dans cette direction, 34 agents thérapeutiques qui, les uns apaisent les échanges gazeux, les autres les accroissent, tandis que d'autres encore n'ont pas d'action ou qu'une action irrégulière.

Nous passerons ainsi successivement en revue le plus grand nombre possible d'agents thérapeutiques, afin de faire le départ de ceux qui exercent sur les échanges respiratoires une action apaisante. Nous présenterons alors un travail d'ensemble de la prophylaxie de la phtisie et des moyens capables

Digitized by Google

de modifier son terrain. Mais, dès maintenant, nous pouvons dire que les grands médicaments traditionnels, comme les arsenicaux, les tanniques, l'huile de foie de morue, par exemple, restreignent les échanges respiratoires. Nous affirmons aussi qu'il est possible de ramener à la normale le chimisme respiratoire exagéré d'un héréditaire, d'un alcoolique ou d'un surmené, par l'association de l'hygiène et des médications restrictives des échanges. Citons trois faits à titre d'exemple :

A. Voici un jeune homme de 19 ans dont la mère et le frère sont morts phtisiques ; la sœur de la mère est morte aussi de phtisie ainsi que ses deux enfants. Ses échanges respiratoires sont exagérés. On le met au repos et à une alimentation d'épargne ; on lui donne de *l'arséniate de soude* à minime dose continue, etc. ; après un an son chimisme respiratoire est redevenu normal :—

	Avant le traitement prophylactique.	Après une année de traitement.
Acide carbonique produit . .	6″·04 .	4″·35
Oxygène consommé total . .	7″·88 .	5″·30
Oxygène absorbé par les tissus	1″·84 .	0″·95

Six ans se sont passés et toute crainte de tuberculose est écartée.

B. Un séminariste de 20 ans, dont la mère, le frère, et la sœur sont morts phtisiques, mais ayant encore deux frères et deux sœurs indemnes, maigrit, pâlit et s'affaiblit. Les échanges sont exagérés. On le met au repos absolu et à la *médication cacodylique* renforcée *d'huile de foie de morue.* En un mois les échanges s'abaissent, tendent à se rapprocher de la normale, pendant que l'état général redevient satisfaisant :—

	Avant le traitement.	Après un mois de traitement.
Acide carbonique produit . .	6″·136 .	4″·647
Oxygène consommé total . .	8″·452 .	7″·077
Oxygène absorbé par les tissus	2″·316 .	2″·120

C. Une jeune fille de 17 ans, avec une mère phtisique et un père mort de granulie, perd l'appétit, maigrit, pâlit et s'affaiblit. On la met au *repos absolu* aidé d'une *médication apéritive* et d'une *alimentation d'épargne.* Son chimisme s'abaisse presque aussitôt :—

	Avant le traitement.	Après le traitement.
Acide carbonique produit . .	5″·760 .	5″·481
Oxygène consommé total . .	7″·927 .	7″·067
Oxygène absorbé par les tissus	2″·160 .	1″·586

Le repos et l'alimentation—nous ne disons pas la suralimentation—ont donc suffi à modérer les échanges respiratoires. En même temps disparaissaient les symptômes inquiétants. Quatre années se sont écoulées ; la jeune fille est mariée, mère de famille et en parfaite santé.

Il y a donc des médications capables de modifier le terrain, et l'examen des échanges respiratoires permet de déterminer ces médications, comme aussi de savoir si elles ont agi dans tel cas particulier, et par conséquent, si tel individu prédisposé a perdu, au moins temporairement, sa prédisposition.

Digitized by Google

4°. Ce *mode de prophylaxie* de la tuberculose qui s'adresse à son terrain mérite d'attirer l'attention au même titre que celui qui consiste à poursuivre le bacille, et la lutte contre la tuberculose ne saurait être efficace sans le concours de ces deux éléments.

Aux mesures d'hygiène publique et privée actuellement édictées contre le bacille de la tuberculose, il faut donc ajouter *l'examen individuel du chimisme respiratoire* de tous les individus soupçonnés de prédisposition. Et de même qu'on vaccine contre la variole et que l'on recherche des vaccins contre d'autres infections, de même il faudra *traiter préventivement*, chez les individus reconnus prédisposés, l'aptitude à contracter la tuberculose, et cela non par des vaccins, mais par les moyens reconnus propres à modifier les conditions chimiques et vitales du terrain ; à savoir l'exagération des échanges respiratoires et la déminéralisation organique qui ferait l'objet d'une étude ultérieure.

Nous vous demandons de vouloir bien mettre cette étude à l'ordre du jour du prochain congrès de la tuberculose, et, dès le mois d'octobre prochain, le laboratoire municipal de thérapeutique de l'hôpital de la Pitié sera à la disposition de tous ceux qui voudrent collaborer avec nous à la définitive mise au point de cette orientation nouvelle de la prophylaxie et peut-être du traitement de la tuberculose.

THE HEREDITARY ARTHRITIC FORMS OF TUBERCULOSIS.

By M. le Dr. G. E. PAPILLON,

Consulting Physician to the Hospital Lariboisière (Paris).

Bearing in mind the "law of pre-tubercular hereditary reaction," which he enunciated and proved at the Thirteenth International Congress (Paris, August, 1900), the author applies that law to arthritic heredity. The existence of an arthritic diathesis, though to some extent disputed to-day, is shown by the hereditary marks it produces, which are bequeathed to the descendants by the victims of any forms of that diathesis: diabetes, gout, rheumatism, asthma, etc. And if, in those descendants, any individual seems, during a part of his life, to have escaped this inheritance, the hereditary tendency will appear under the influence of any slow and hidden infection, as, for instance, the pre-tubercular infection.

In heredo-arthritic people, the pre-tubercular stage may be indicated by any arthritic manifestations, such as fits of asthma, attacks of subacute rheumatism, late appearance of what the French call "stigmates de l'arthritisme." These are not manifestations of localised tubercles, as in the case of the "tuberculous rheumatism" described ten years ago by Poncet, but only effects of the bacillary toxins.

These fits of asthma, rheumatic attacks, arthritic deviations, etc., are the symptoms of the disordered function: either of the contractility of the

Digitized by Google

bronchial muscles, or of the secretion of the articular synovia, or of the nutrition of the extremities of the bone, cartilages and periarticular muscles; disorders all belonging to the great class of "sympathetic reactions to the tuberculous intoxication," which was the subject of a paper read by Dr. Papillon to the Congress of Naples (April, 1900).

The author proves that the toxins introduced into the organism by a tuberculous centre may produce the "stigmates de l'arthritisme" in predisposed individuals, as in others (otherwise predisposed by a different heredity), they may create Papillon's "Pre-tuberculous neurasthænia," or pre-tuberculous anæmia, etc.

PARA-TUBERCULOUS HEREDITARY DYSTROPHY.

By Dr. E. MOSNY, *Hospital Physician, Paris.*

Clinical study and experimental medicine demonstrate the fact that subjects of phthisis do not transmit the bacillus of tuberculosis to their offspring (*typical conceptional heredity*), and that the very rare cases of *congenital tuberculosis* are due to intra-uterine contamination of the fœtus by the tuberculous mother.

Clinical observation, moreover, goes to prove that subjects of phthisis do not transmit to their offspring either a specific predisposition to bacillary contamination or any marked degree of immunity (*a typical specific heredity*). But we should not, however, conclude from this that tuberculosis of parents exercises no influence on the health of their offspring.

Both clinical and experimental medicine, indeed, agree in demonstrating that the tuberculosis of parents has an influence on fecundity and the products of conception similar to that which is exercised by the majority of chronic intoxications such as alcoholism, saturnism, or the infections the evolution of which is slow or prolonged—*e.g.* syphilis (*indifferent atypical heredity*). These consequences, early or late, of the tuberculosis of parents on their future descendants, may be conveniently grouped under the title *para-tuberculous hereditary dystrophy.*

Their frequency is a witness to the grave evil of tuberculosis from a social point of view, which compromises not only the existence of the individual it attacks, but also involves the future, affecting posterity with an irrevocable and permanent taint.

Tuberculosis may even influence conception by producing sterility in the subjects attacked. It may also exercise on the product of conception, from the time of its origin or during the course of pregnancy, either a general influence or one localised more precisely in one of the organs of the embryo or fœtus.

Generalised manifestations of hereditary dystrophy appear at all stages of its existence; from conception, or during the course of pregnancy, they are shown by sterility, death of the fœtus, abortion, premature birth; when

Digitized by Google

pregnancy arrives at its normal limit, by still-birth, weakness of the offspring, which is reared badly, pines away, and dies prematurely without adequate cause, or, indeed, survives in a piteous condition, badly or incompletely developed (infantilism, juvenilism, dwarfism), a fit subject for all forms of decay, and exposed without defence to all kinds of contamination. At other times the influence of the tuberculosis of parents on the health of their offspring localises itself, or shows itself under multiple forms of most varied manifestation, the consequences of arrest or perversion of normal development.

Among these localised manifestations of para-tuberculous dystrophy the following appear with a frequency that is very marked: hypoplasias of the vessels and blood, pure mitral stenosis, stenosis of the pulmonary artery, generalised stenosis of the arteries (arterial aplasia), and chlorosis.

Generalised or localised manifestations of para-tuberculous hereditary dystrophy, and the functional troubles which sometimes depend on them, become apparent, either at birth or not till later on at one of the important stages of growth—*e.g.* weaning, dentition, puberty. Tuberculosis of the father or that of the mother—above all, the latter—is the determining course of these dystrophic congenital defects. Tuberculosis of the parents no more predisposes the offspring to bacillary contamination than it confers immunity against attack. But the offspring of phthisical subjects, organically and functionally imperfect, weak from the time of birth, and more tried than others by the vicissitudes of growth—seem to be predisposed to bacillary infection, because the risks of contagion are multiplied infinitely by contact, immediate and continuous, with contaminated parents, and because their weakened frame offers less resistance to the infection so produced. The apparent immunity of some descendants of tubercular subjects who carry with them certain manifestations of para-tuberculous hereditary dystrophy is, in reality, the consequence of the very nature of the dystrophic lesion or of its localisation, and not the manifestation of a real condition of resistance. Such is the *relative immunity* in regard to tuberculosis exhibited by the subjects of chlorosis or pure mitral stenosis.

L'HÉRÉDO-DYSTROPHIE PARATUBERCULEUSE.

PAR M. LE DR. ERNEST MOSNY, *Paris.*

LA tuberculose peut influencer très diversement la descendance des sujets qui en sont atteints.

Parfois, les enfants des phtisiques sont infectés avant de naître; mais jamais l'infection n'est d'origine conceptionnelle. Il s'agit toujours alors d'infection maternelle transmise au rejeton par la circulation placentaire: c'est une véritable contagion intra-utérine. Cette forme d'hérédité de la tuberculose, désignée sous les noms d'hérédité du germe, d'hérédité directe, typique, similaire, ou homœomorphe, est exceptionnelle; et l'on peut dire qu'en

Digitized by Google

réalité, la contagion post partum résume presque à elle seule toute l'étiologie de la tuberculose.

La tuberculose des parents ne laisse pourtant pas que de déterminer des modifications multiples et variées de la constitution de leurs enfants, modifications qui sont considérées comme les manifestations d'une hérédité de terrain, hérédité indirecte, atypique, dissemblable ou hétéromorphe.

Il y a deux modalités possibles de l'hérédité atypique : —

1°. Une forme spécifique qui, chez les rejetons de souche tuberculeuse, pourrait se présenter sous les manifestations opposées de la prédisposition ou de l'immunité, et qui serait caracterisée dans le premier cas par une réceptivité particulière à l'égard de l'infection tuberculeuse ; dans le second cas par une constitution plus ou moins réfractaire à ses atteintes.

Ni la clinique ni l'expérimentation ne nous donnent des preuves suffisantes, indiscutables, de l'existence, chez les enfants des phtisiques, de l'une ou de l'autre de ces deux modalités opposées de l'hérédité atypique spécifique.

2°. Une forme indifférente, reconnaissant pour cause des modifications cellulaires dûes à l'action de l'infection tuberculeuse, influençant soit l'ovule ou le spermatoblaste avant la conception, soit l'œuf pendant la gestation et comportant les insuffisances fonctionnelles et les malformations organiques les plus variées.

Ce sont ces faits en apparence disparates, mais en réalité reliés entre eux par une pathogénie commune, que j'ai proposé de grouper sous la dénomination d'hérédo-dystrophie paratuberculeuse,* voulant indiquer par là que si la tuberculose en est une excuse fréquente chez les rejetons des parents qui en sont atteints, elle n'en est pas la seule. Infections ou intoxications lentes et prolongées comme elle peuvent, au même titre et par le même processus, en être également l'origine : telles sont la syphilis, l'alcoolisme, le saturnisme, pour ne citer que les plus fréquentes.

Les manifestations de l'hérédo-dystrophie paratuberculeuse sont nombreuses et variées : l'une de mes élèves, Mlle H. Kowner, en a, dans sa thèse récente, relaté maints exemples divers.

C'est qu'en effet, la tuberculose peut influencer de maintes façons différentes le produit de la conception. Elle peut manifester son action, avant même la conception, par l'incapacité du germe mâle ou femelle à la fécondation ; stérilité ; pendant la gestation, par l'incapacité du germe fécondé à la maturation ; avortements, accouchements prématurés ; à la naissance enfin, par l'incapacité du fœtus à la vie : morti-natalité.

La grossesse parvient-elle à terme, cette débilité congénitale va se révéler, dès la naissance, par l'incapacité du nouveau-né au développement normal, incapacité qui s'accuse plus évidente et plus menaçante à chacune des grandes étapes de la croissance : au moment du sevrage et de la première dentition, au moment de la seconde dentition, à la période prépubère, à la puberté.

---

* *E. Mosny.*—"La descendance des tuberculeux. Manifestations spécifiques et indifférentes de l'hérédité atypique de la tuberculose. Hérédo-dystrophie paratuberculeuse" ('Revue de la tuberculose,' 1901, No. 3).

Digitized by Google

Le nouveau-né chétif, de moindre poids, s'élève mal, dépérit sans cause appréciable, et succombe parfois sans motif apparent, d'autres fois à l'occasion de la dentition ou du sevrage, d'autres fois encore aux atteintes d'une infection quelconque souvent banale et bénigne en apparance, à laquelle sa débilité congénitale ne lui permet pas de résister. Ainsi voit-on fréquemment, dans certains ménages tuberculeux où l'un des parents ou bien les deux à la fois sont atteints de tuberculose, se succéder les fausses-couches, la mort prématurée des nouveau-nés, ou celle des enfants dans le cours des deux premières années. Ainsi souvent s'éteignent, par une véritable sélection naturelle, nombre de familles tuberculeuses dont aucun des enfants ne peut atteindre le terme de la deuxième année.

Lorsque l'enfant chétif, de souche tuberculeuse, a pu survivre à ces premiers assauts, lorsqu'en dépit d'une croissance anormale qui s'est annoncée telle dès le début de la vie, il peut péniblement atteindre l'âge de la puberté, à travers mille entraves, et au prix de mille accrocs dont chaque étape de la croissance a subi l'influence, et laissera subsister l'empreinte, souvent alors on verra plus manifestement éclater, à l'occasion de la puberté ou de la période qui la précède, la débilité qui généralement le livre sans défense à la contagion familiale de la tuberculose.

À chacune des étapes de la croissance le développement hésite, s'arrête ou dévie du droit chemin, trahissant par des malformations diverses son impuissance ou ses erreurs, accusant plus profondément la débilité organique et l'insuffisance fonctionnelle de l'être déchu dès sa procréation et laissant persister une empreinte indélébile qui sera, chez l'adulte, sa marque d'origine.

Tel est l'habitus tuberculeux, qui n'est que la conséquence d'un retard ou d'une série d'arrêts du développement.

Il est aisé de prévoir quel peut être l'avenir de ces êtres chétifs nés et élevés dans une famille tuberculeuse où les risques de contagion se trouvent accrus du fait même de la cohabitation et du contact incessant des parents contaminés.

Quelque fréquente que soit cette forme généralisée de l'hérédo-dystrophie paratuberculeuse qui se présente sous les aspects les plus variés de la débilité congénitale, il s'en faut que ce soit là la seule manifestation de l'influence que peut avoir la tuberculose des parents sur le développement de leurs rejetons.

Souvent en effet cette influence se précise, les perversions du développement se localisent et se présentent sous les formes multiples des malformations les plus variées.

Tantôt alors ces malformations profondes, disséminées, constituent de véritables monstruosités incompatibles avec la vie. Tantôt et plus souvent il s'agit de malformations plus limitées, plus localisées, dont la gravité dépend de l'importance de l'organe ou des tissus qui en sont atteints. Malformations osseuses du rachitisme, malformations dentaires, pied-bot, bec-de-lièvre, perversions fonctionnelles, et troubles dynamiques du système nerveux, ont tour à tour été considérés comme des manifestations possibles de l'hérédité atypique de la tuberculose. On peut même dire qu'il n'est guère de malformation congénitale qui n'ait été ou ne puisse être un jour attribuée à la tuberculose des parents.

Digitized by Google

Toutefois, il s'agit là de malformations si banales, si communément attribuées avec une égale probabilité à l'alcoolisme, à la syphilis, et en général à toutes les infections ou intoxications chroniques des ascendants, que leur étiologie réclame de nouvelles études, et qu'il n'y a là rien, en tous cas, de spécial à la tuberculose.

Mais il y a telles autres manifestations dystrophiques de l'hérédo-tuberculose atypique qui, sans être rigoureusement spécifiques, sont néanmoins si fréquentes chez les rejetons des tuberculeux qu'on les a longtemps considérées comme des manifestations d'une lésion bacillaire congénitale, dont l'évolution fibreuse aurait effacé les caractères spécifiques au point de rendre méconnaissable leur origine et leur nature tuberculeuses.

C'est qu'en effet la tuberculose, comme la syphilis, a ses préférences ; comme celle-ci, elle choisit les tissus sur lesquels elle évolue, les voies par lesquelles elle se propage, les organes où elle se fixe. De même aussi, l'hérédo-tuberculose, comme l'hérédo-syphilis, a ses stigmates dystrophiques de prédilection qui sont, au premier chef, les hypoplasies angio-hématiques : le rétrécissement mitral pur, le rétrécissement de l'artère pulmonaire, le rétrécissement généralisé des artères, la chlorose en sont les manifestations les plus communes.

Le rétrécissement mitral pur s'observe fréquemment chez les descendants des tuberculeux ainsi que l'ont signalé MM. Potain et Teissier, ainsi que j'en ai observé des cas rapportés dans la thèse de Mlle Kowner. Non moins souvent que le rétrécissement mitral pur, le rétrécissement congénital de l'artère pulmonaire s'observe chez les rejetons des phtisiques : Hanot et moi-même en avons relaté d'assez nombreux exemples.

Dans les cas de rétrécissement congenital, généralisé des artères, ou aplasie artérielle généralisée, où les antécédents des malades ont été soigneusement relevés, on trouve la tuberculose des parents ; et j'ai récemment observé, chez un jeune homme de souche tuberculeuse, l'existence simultanée d'un rétrécissement mitral pur, d'un rétrécissement de l'orifice aortique, et d'une aplasie artérielle généralisée, toutes lésions congénitales qu'il convenait, sans aucun doute, d'attribuer à l'influence de la phtisie maternelle.

Si, de ces faits, on rapproche la constatation, maintes fois répétée, de la chlorose chez les filles des tuberculeux, et la fréquence, chez les chlorotiques, des hypoplasies cardiaques et vasculaires que je viens d'énumérer, on verra qu'une étiologie commune, la tuberculose des parents, établit outre toutes ces affections congénitales, malades d'evolution, un lien qui les unit indissolublement entre elles, et permet de superposer, à l'étiologie commune, une pathogénie commune. À l'origine de toutes ces affections se place la tuberculose des ascendants, déterminant chez leurs enfants, entre autre malformations congénitales, mais avec une fréquence toute spéciale, des hypoplasies cardiaques, vasculaires ou hématiques, que la pathologie décrit sous les noms de rétrécissement mitral pur, de rétrécissement de l'artère pulmonaire, de d'aplasie artérielle généralisée, de chlorose, dont les formes pures ou associées constituent les manifestations les plus fréquentes de l'hérédo-dystrophie paratuberculeuse.

Ce n'est pas seulement la tuberculose de la mère qu'il faut incriminer dans

Digitized by Google

la pathogénie de ces malformations, c'est aussi celle du père, ainsi que maints exemples en font foi. Toutefois, il est indéniable que la contamination maternelle a plus d'influence que la contamination paternelle sur la constitution des rejetons : c'est qu'en effet, la débilité du germe se trouve accrue pendant la gestation par l'action incessante des toxines circulant dans le sang maternel.

Qu'il s'agisse de dystrophies générales (infantilism, juvénilisme, nanisme) ou de malformations partielles (hypoplasies cardiaques, artérielles, hématiques) ; l'origine congénitale parfois même conceptionelle de ces lésions organiques ne saurait être démentie par le fait, si souvent constaté, de l'apparition plus ou moins tardive des troubles fonctionnels qu'elles provoquent. Débilité organique, malformations viscérales, hypoplasies variées, peuvent être méconnues à la naissance, demeurer plus ou moins longtemps latents ; elles n'en existent pas moins en puissance. Tôt ou tard, l'influence fonctionnelle qu'elles comportent éclatera lorsque l'une des grandes étapes de la croissance et surtout l'échéance de la puberté mettront l'organisme en demeure de faire face au surcroît de vie plastique que lui imposent, et la suractivité soudaine du développement, et la rupture momentanée de l'équilibre normal entre les divers segments du corps, les organes et leurs fonctions.

Aussi bien est-ce à l'époque de la puberté que se révélent brusquement sans cause appréciable, parfois à l'occasion d'un accident insignifiant, les troubles fonctionnels révélateurs des hypoplasies cardiaques, vasculaires ou hématiques, toutes lésions congénitales dont l'existence avait jusqu'alors passé inaperçue : la chlorose en est le meilleur exemple.

C'est d'ailleurs souvent aussi à l'époque de la puberté qu'apparaissent chez les descendants des tuberculeux, atteints ou non de tares dystrophiques, les premiers symptômes de l'infection bacillaire.

Les rapports des manifestations diverses de l'hérédo-dystrophie paratuberculeuse avec l'éclosion de la tuberculose ont été très diversement appréciés, puisque l'on voit l'opinion osciller successivement de la prédisposition à l'immunité.

Il semble en réalité que si les tuberculeux font souvent souche de dégénérés, d'êtres organiquement et fonctionnellement imparfaits, ils ne transmettent guère à leurs enfants, du fait de leur tuberculose, ni prédisposition ni immunités spécifiques, à l'égard de la contamination bacillaire. Mais il n'en demeure pas moins évident que, selon leur nature ou leur localisation, certaines manifestations de l'hérédo-dystrophie paratuberculeuse, telles que la constitution débile, infantile ou juvénile, l'étroitesse du thorax, favorisent le développement de l'infection bacillaire contractée dans le milieu familial, tandis que certaines malformations congénitales précoses ou tardives, telles que les hypoplasies cardiaques, vasculaires ou hématiques, que nous avons citées, semblent au contraire opposer à son évolution des conditions capables d'en arrêter le progrès.

L'étude minutieuse, constante et longtemps poursuivie des familles de tuberculeux nous démontre généralement d'une façon fort nette la coïncidence de l'apparition des dystrophies congénitales dans la ligne des descendants, avec

Digitized by Google

l'époque du début présumé de la tuberculose des parents. Toutefois il s'en faut qu'il en soit toujours ainsi ; souvent même, à côté d'enfants sains et vigoureux, entremêlés sans ordre dans la ligne des descendants, naissent des enfants qui viennent au monde avant terme, succombent à la naissance ou en bas âge, naissent et vivent avec des malformations variées, ou restent des déchus, des dégénérés, des infantiles. Il semble alors que le réveil ou l'activité momentané de lésions éteintes ou habituellement silencieuses, chez les parents, aient, avec l'apparition des tares dystrophiques chez les rejetons, des rapports de simultanéité qui entraînent presque fatalement l'idée de causalité.

Tous les faits qui précèdent, et dont je n'ai pu présenter ici qu'une esquisse rapide, mettent hors de contestation l'influence dystrophiante du virus tuberculeux, et peut-être aussi celle des produits modifiés des cellules des parents contaminés sur le produit de la conception. L'expérimentation a pu reproduire sinon dans le détail, du moins dans leurs grandes lignes, les manifestations les plus générales de l'hérédo-dystrophie paratuberculeuse. L'embryologie et la tératologie expérimentales nous expliquent tous ces faits, et nous montrent par maints exemples que les conditions anormales de tous ordres où se trouvent la cellule sexuelle avant la fécondation ou bien l'œuf fécondé depuis le début de la segmentation, et pendant tout le cours de la gestation, ou de l'incubation, sont les conditions déterminantes des troubles de l'ontogenèse, des arrêts ou des perversions du développement.

Les manifestations multiples et variées de l'hérédo-dystrophie paratuberculeuse qui ne sont qu'une confirmation, pour un cas spécial, de cette loi générale, nous révèlent la gravité sociale de la tuberculose, qui ne compromet pas seulement l'existence de l'individu qu'elle frappe, mais qui engage l'avenir, parce qu'elle marque irrévocablement la descendance de tares indélébiles, parce qu'elle fait généralement souche de déchus, inaptes sinon toujours à la vie, du moins aux obligations qu'elle lui impose, et préparés à toutes les contagions.

DE LA TRANSFORMATION DU TERRAIN TUBERCULEUX HYPOACIDE EN TERRAIN ARTHRITIQUE HYPERACIDE.

PAR M. LE DR. SAMUEL BERNHEIM, *Paris.*

L'AUTEUR commence par étudier le caractère du terrain tuberculeux dont l'état bio-chimique doit être bien connu de tout le monde aujourd'hui. La mesure de la minéralisation et de l'état chimique de l'organisme peut en quelque sorte être la mesure de sa résistance. Or on sait que le tuberculeux possède un terrain déminéralisé, déchloruré, déphosphatisé et surtout hypoacide, tandis que l'arthritique a un sol surminéralisé, très riche en chlorures et hyperacide. C'est à dire que par leur formule chimique ces deux terrains sont l'inverse l'un de l'autre. De même cliniquement il existe une

Digitized by Google

sorte d'antagonisme entre la tuberculose et la diathèse arthritique : la phtisie est rare chez les arthritiques et bénigne chez eux. De ces premières données chimiques, biologiques et cliniques, l'auteur tire de nombreuses déductions thérapeutiques.

C'est ainsi que, suivant lui, le régime des sanatoria, le repos absolu, la suralimentation, la cure d'altitude n'ont d'autre but et d'autres effets que la transformation du terrain. Tous ces facteurs bienfaisants, qui agissent moins sur le bacille que sur l'organisme, tendent à augmenter l'acidité organique du malade. Et de fait, l'auteur a soumis au repos complet un certain nombre de sujets et il a constaté chez tous, au bout d'un certain temps, une augmentation très sensible de l'acidité urinaire. De même la zomothérapie si recommandée par MM. Richet et Héricourt n'agit pas par les antitoxines, mais par une transformation du terrain dont ils enrichissent l'acidité.

Après avoir donné ainsi interprétation des faits, M. Bernheim ajoute qu'il est possible d'obtenir cette transformation du terrain, surtout chez le tuberculeux soigné précocement par un régime hygiénique et diététique, et en saturant l'organisme avec un acide puissant tel que le phosphate de créosote ou l'acide phosphorique. Dans les Dispensaires antituberculeux de Paris, il a observé un grand nombre de phtisiques dont il a dosé fréquemment l'acidité urinaire. Or il a constaté que sous l'influence du phosphate de créosote injecté à la dose de 3 grammes tous les 2 jours, l'acidité augmentait rapidement et l'état général améliorait. D'où on est en droit de conclure qu'il est possible de transformer artificiellement un terrain hypoacide favorable à l'évolution tuberculeuse en un terrain hyperacide antagoniste du bacille de Koch. Cette transformation, qui est, suivant l'auteur, la planche de salut du phtisique, doit être recherchée et peut être obtenue par un régime hygiénique et diététique et aussi par des moyens thérapeutiques très actifs.

Digitized by Google

Digitized by Google

DISCUSSION ON SANATORIUM TREATMENT.

OPENING ADDRESSES.

I.—By T. Clifford Allbutt, M.D., LL.D., D.Sc., F.R.S., F.R.C.P. Lond. and Irel.,

Regius Professor of Physic, University of Cambridge; Physician to Addenbrooke's Hospital, Cambridge, England.

Ladies and Gentlemen,—

It is with diffidence that I stand forward to address you to-day, seeing that many of you are occupied in the work of the sanatorium. It seemed well to the officers of this section of the Congress that this subject should be introduced by one standing outside the sanatorium, who nevertheless had been concerned with the treatment of tuberculosis in all its fashions for a long series of years, and in a position thus to estimate the ultimate results of treatment of various kinds and in various places. I cannot forbear from expressing my regret that this duty was not undertaken by our distinguished friend, Sir Hermann Weber.

My interest in this subject was first aroused by Dr. Archibald Smith, of Lima, with whom I had some acquaintance in the "eighteen-sixties." He spoke, for the first time in my experience, with confidence of the curability of the disease, especially as treated in the mountains of Peru. These new hopes were fortified by Henry Bennet, with whose methods and results I became familiar, both in his writings and in personal association with him at home and in the Riviera. The first records, of any considerable value, of the effects of various methods of treatment of the disease were published by Dr. C. J. Williams and Dr. Theodore Williams in 1871, records which did much to instruct and to encourage us. Bennet's influence drew me away for a time from the High Alps as a climate for phthisis, but in 1878 I, after occasional observation of a few cases treated in the Swiss Alps, made the acquaintance of Dr. Unger and Dr. Ruedi, and I then formed great anticipations of the value of Alpine climate—anticipations which twenty-five subsequent years have strengthened and formulated.

At that time climate seemed to us to be the paramount factor in the cure; so that our admiration was the greater as the remarkable results obtained by Brehmer at Görbersdorf rose into repute, results in which climate played but a secondary part. Brehmer's method, to which nothing important has been added by later physicians, brought the cure of phthisis from a remote and costly achievement, attainable only by wealthy and leisurely patients, near the doors, I had almost said into the homes, of poorer sufferers given up to death.

Digitized by Google

We did not realise the facts which the computations of Birch-Hirschfield and others have placed beyond question, that then, as now, many persons were recovering from pulmonary phthisis under our eyes; our eyes were not open to see it.

By the modern method we are not only curing the individual, but we are also laying the chief stone of the edifice of prevention. This modern method we may call the method of Brehmer; but it is called more commonly the Sanatorium method. To this name some objection has been made; it is said that the method may be carried out independently of the sanatorium. We cannot be guided by the etymology of names—no name can indicate all the connotations of its subject; but I think that this objection is a shallow one. The method was worked out in a sanatorium created for the purpose, it has been tested and perfected in sanatoriums, and if now it can be carried on outside a sanatorium, which is not too readily to be admitted, this is so only by bringing the skill and the conditions of the sanatorium into the home. I should look with little confidence on the home treatment of a phthisical patient by a physician unversed in the practice of the sanatorium, and by a patient who had not had at least a month's training within its precincts. It is, as it were, to tell a boy at home to read his Cæsar and his Xenophon, a little Euclid and algebra, to pat him on the head, bid him be good and industrious, and promise to call in a week or ten days to see how he has got on.

It is no paradox to say that in the congregations of the sanatorium we have learnt individual treatment; that from institutions which may well sink into the thraldom of routine, or lend themselves to the grosser temptations of the hotel-keeper, has issued the doctrine that for the individual sufferer no vigilance is fastidious, no skilled control vexatious. From a highly specialised system we have learnt that for phthisis there is no specific—not even climate; that to speak of the best climate for phthisis, of the best mode of feeding, or again of the best disposal of rest and exercise, is to speak vainly. For scrofulous children, as a rule, the best climate is the sea; for the adult in the third or fourth decade of life the climate of the high Alps, less windy than the sea, even more tonic and perhaps having some virtue in its dryness and rarity, is often the best; the elderly must be content with a milder and more equable resort, which indeed is to be preferred also for many younger patients whose stomachs are unequal to large demands, and whose heat production is slow. On the robuster folk full feeding may be pressed quickly; with the enfeebled, whose stomachs are often relaxed, large and rich meals best agree when they are vomited. To the febrile and overwrought again that prolonged rest, which in vigorous and apyretic persons would be lost time, is precious.

The sanatorium, if on the block system, should stand south-east, on a dry upland site sheltered by pine woods, 200 yards at least away from the building. The front should be slightly crescentic. Most of the rooms should be single, a few may be double for special needs. All must be on the front, the back being given to the service, and to the accommodation of an

Digitized by Google

occasional visitor. The smaller the bedroom, within limits, the better, lest it be used as a sitting room, whereas it is but a shelter for the bed, and for dressing and undressing. There should be no general sitting rooms, which encourage indoor gossip, except, of course, a dining room and a spacious and airy entrance hall on the south front. Every room should have its covered balcony, wide enough to take the long chair (or bed) and a table, separated by glazed screens from those next to it, and supplied with convenient electric light. The balcony is the living room of the patient. Airing galleries may be provided for the few who may like them, but they have social and other disadvantages. The balcony, if raised a step or two above the bedroom floor, will not shut out much light from the window below—at any rate, not in winter, when the sun is low. Of walls and furniture I will only say that the doors should be double, and the walls and floors without angles and well pugged; that wardrobes should be built in the walls, flush with them and the ceiling; that linoleum, with felt under it, makes the most comfortable and the cleanest carpet. The window must occupy the greater part of the south wall, and sliding shutters with louvres must be provided to darken the room for light sleepers, and against stormy weather. Heating is a great difficulty. Open fires, if practicable, are by far the best means of heating. To heat the air to be respired is wrong, both in principle and in practice. If so-called "radiators" must be used, the radiator must be set against an opening in the outer wall, and the air led over it into the room, so as to enter the room at the bottom. Hot air rises, and, do as we may, the inmate of a room so heated is hot in the head and cold in the legs and feet. The roof must be warm in winter and cool in summer. If all passages and corridors be well and equally warmed, the heating of the chambers will need less attention.

It is undesirable that patients should take their principal meals in the private room, and a dining hall must be provided. But I have received a bad impression of the dining rooms of most of the sanatoriums which I have visited. Either they are a whirlpool of draughts, wherein the poor patients can scarcely keep their hair on, or they reek with foul odours. The director of a sanatorium, in many ways admirable, lately ushered me with pride into a handsome dining hall whose close atmosphere was so thick with the reek of departed dinners that I sickened on the threshold and, muttering some excuse, made my escape. Most of these halls are much too high, and not thoroughly air-swept; others are ordinary apartments with the windows taken out, so that the patient must dine in a hurricane, bolting his food and surrendering whatsoever charm dinner might yet have for him. Saloons should give place to sheds or bungalows with sliding walls of louvred shutters which can be adapted to the wind of the day, and afterwards quickly thrown open for coffee and cigarettes, when each patient can sit where he pleases.

If, in obedience to the needs of municipal or other public bodies, such sanatoriums must be built to receive more than fifty patients, a physician must be engaged for every multiple of fifty—the largest number of patients to which any man can do well. Let us not tolerate again the farce of so-called

Digitized by Google

medical attendance which still persists in many asylums for the insane; to the grievous loss of science and to the detriment of the sick. But in my opinion sanatoriums are designed so large and costly as to deter the benevolent and to encumber the treatment. A hut or villa system in the grounds about a central administrative block is cheaper, more homely, and more efficient. The Japanese house is a good model. The difficulty of ventilating large blocks is insuperable; they will beget the secretary-director, and lead to a barrack or hotel system. Tents are useful, but for temporary purposes only. Ordinary dwelling-houses converted into sanatoriums are makeshifts at best, and unsatisfactory at that.

I repeat that it is vain to talk of a "best climate" for phthisis, even for those who can go where they please. Sun is an amenity and a cordial, but does not directly contribute much to the cure. At Davos the patients do as well in the darker as in brighter winters. Gabrilowitch, at Halila, compared the reports for the winter six months with those of the summer six months, and found that the winter patients did considerably better than the summer patients, the bracing air of winter, in spite of less light, being more restorative. For the most general terms in which climatic conditions can be put is that the coldest air which the individual can tolerate, if it be dry, clear, and still, is the best, as it calls for more food and thus stimulates the appetite. But in an air so cold as to be very stimulating to a patient of thirty-five years of age, a patient of fifty-five years of age would shrivel up. Thus it is for the younger patients of fair vigour that the high Alpine airs are the best, say for vigorous persons under forty or forty-five years of age without much bronchital irritation. At Davos it is wonderful to see a wan listless youth for the first few days picking daintily at his half-eaten meals, and ten days later devouring all before him. At lower altitudes some urgent feeding may be needed, but in any fresh open air a naturally good feeder soon regains appetite; and to force him before Nature calls is too often to lose a week or two in an attack of indigestion. In England, Norfolk, Suffolk, and Kent offer the best climates for the phthisical. A poor eater does badly in any climate and under any stuffing. Appetite again much depends upon the preparation of the food, especially among the wealthier classes. The meals, even in the more luxurious sanatoriums, are too often badly cooked and badly served, good as the raw materials usually are. The dishes and plates are cold, the meats are tepid, and the coffee and tea are scarcely fit to drink. All bread, rolls, and cakes should be baked in the house. A variety of dishes, again, as city diners know, tempts to repletion; yet sanatorium cooks do not stand alone in their contentment with a narrow round of menus, which, attractive enough at first, begin, perhaps after a week or two, to pall upon the palate. The physician of a sanatorium should be something of a cook and much of a gourmet.

For the encouragement of scientific work in sanatoriums, which in the nature of the case are far from the advantages of large towns, there should be central committees. Such a committee in England—let us say, a branch of the National Association for the Prevention of Consumption and other

Digitized by Google

Forms of Tuberculosis—would organise the methods of reporting, would suggest the kind of information needed, would circulate scientific results among these institutions, and would point out the many directions in which investigation is needed. Excellent work of this kind is now done in a few sanatoriums; but there is a fear lest the medical officers should fall into the supine routine of too many of the asylums for the insane. Or, again, work may be active but ill-directed. Every sanatorium deserving the name has its laboratory; but how much valuable time is wasted in the counting of bacilli? Such computations are necessary from time to time, no doubt; but daily or even weekly fluctuations are too dependent on temporary contingencies to be worth much, and there are more important things to be done. For instance, I may suggest examinations of the blood, the comparison of its cells, and of the fluctuations of its specific gravity; the study of its sera for agglutination, toxins and antidotes, and their relation to the tuberculins; the attenuations of virus by inoculation and other methods; the estimation of the virulence rather than of the number of bacilli in the sputa; the evidences of mixed infections such as cocci in the blood; the investigation of the possible harm of re-infection by tubercle, as, for example, by soiled fingers, tooth-brush, or swallowed sputum; the secretions of the stomach at various phases of digestion; examinations of the urine for toxins, for the comparison of ingestion and excretion, for the influence of fever and of exercise on metabolism; and so forth, *ad infinitum*. Many a worker on such subjects in a remote sanatorium would feel the moral tonic of the help and association of a central scientific committee. Again, in all public or semi-public institutions there should be a bench in the laboratory where any pathologist, neighbour or visitor, could avail himself of the material either for research or for self-improvement. I attach importance also to the establishment of some connection between the District Medical Officers of Health and the sanatoriums; but this is a long story.

I am guilty of no extravagance when I suggest that one-third of you who hear me, wittingly or unwittingly, are, or have been, infected with tubercle. Some of us, ailing indefinitely, may have been the innocent means of infection to others; so secretly stalks infection in our midst. Every trustworthy report from sanatoriums—and herein I must compliment our colleagues of Germany on such reports, the last in my hands, and this not the least, being that from the sanatoriums of the Hanse Towns—every such report, I say, emphasises anew the remarkable success of Brehmer's system, on one condition—namely, that the patient be caught at the outset of his disease. Happily for me, it is not within the sphere of this paper to ask what provision is to be made for advanced cases, whether for their own comfort or for the safety of the public—this is a matter of State Medicine; but it is my duty to reinforce your demand that advanced cases, save in a few overwhelming infections, shall cease to occur. There has been much supineness in the matter of early diagnosis—a supineness bred of pessimism, of despair; let us bring the inspiring message of optimism, of enthusiasm. Let every physician, however modest his sphere, remember that upon his alertness

Digitized by Google

depend the lives of the infected and the stamping out of infection. The incipient case of to-day is the advanced case of to-morrow. On former occasions I have said that a case presenting the ordinary first stage symptoms of the out-patient room, if for our fathers an incipient case, is in our eyes an advanced case; yet too often still the family practitioner waits till the physical signs are evident to a second year's student. What consultant has not felt his heart sink as, in the so-called incipient case, the "consonating râle" fell upon his ear? Medical men who have no ear for music ought to distrust themselves a little in auscultation. Not long ago a physician, as honest as he is accomplished, told me that he could not perceive a difference of percussion which I estimated at quite a quarter of a note. He lamented his lack of an ear for music. Early diagnosis depends, however, less upon this detail or that, more upon the cumulative evidence of many indications which the family physician is in the best position to appreciate. The features of the individual do not carry much weight with me, excepting, of course, such acquired features as scars in the neck, or the thick nostrils and upper lip due to the catarrhs of childhood. Many persons of so-called tuberculous aspect never fall victims to the bacillus; many of robust appearance are attacked. Never let muscular strength, ruddy cheeks, or a well-formed chest blind us to canker within.

Whosoever complains of being overwrought, of being "off colour," as the phrase goes, in him suspect phthisis, cough or no cough. Let no hæmoptysis, however slight, be set down to a "blood-vessel in the throat"; let no pleurisy, however long ago, be forgotten. In examining the sputum, if any there be, we shall not take the absence of bacilli as an assurance of safety. In young children we may find some occasion of examining vomit for bacilli. If there be no fever, a high mean pulse-rate with low arterial pressure will keep us on the watch, especially if therewith there be some loss of weight, an unusual tendency to sweat and languor after exertion. Indeed, we shall not admit the absence of fever until we have taken the temperature in the rectum every two hours of the twelve of the day, and particularly after exercise. To anæmia, to dyspepsia, to vague pains in the chest, if we shall not give more than their due weight, we must not give anything less. Percussion, which must be made on the bare chest of the patient, sitting on a wooden chair without cushion, must be made with a light finger, and the notes must be estimated not so much by absolute pitch as by an attentive comparison of the two sides. Even if both apices be affected, the notes of the two are never identical. I would ask you if comparison of the percussion notes in inspiration and in expiration is of importance? The larynx should always be examined, if only for loss of colour. The Röntgen rays, so anxiously tested by Dr. Walsham and others, have not yet proved to be of use in doubtful cases. The spirometer, valuable for comparative records in the various stages of a patient under treatment, is of no use in early diagnosis. Attempts to plot out curves of fever characteristic of tubercle or mixed infections seem not yet to have met with much clinical success. Of tuberculin in diagnosis I have had little experience—in one case, however, it was a great help to me—yet tubercle sleeping in many of us, if not by

Digitized by Google

its use awakened for mischief, might give some ominous answer in its dreams.

The next question I would submit for consideration is that of the mean duration of residence in the sanatorium. The degrees of cure are three—namely, arrest, obsolescence, and *restitutio ad integrum*. To consider this last is to be too curious. Nay, even if we make obsolescence our term for discharge, we shall nip sanatorium treatment in the bud—at any rate, for the poorer classes. To bring about obsolescence of phthisis, even of the first stage, we need, in my opinion, two winters and one summer at least—say, eighteen months—and in saying this I am astonished at my own moderation. In many cases three winters and two summers will be needed. But how many, even in the easier classes, can sacrifice this time without breaking up their careers and abandoning the ties of home? Such, nevertheless, is the advice which for many years I have had to offer to those who have consulted me; and this opinion I must still give if we are to be satisfied with nothing short of obsolescence. But during the last few years, during which time statistics, such as those of Dr. Turban and of Dr. Trudeau, have been given to the world—since we have had to reckon with what is practically possible in the provision of sanatorium accommodation, and to consider if it be worth while to spend money on a vast scale for cures beyond our compass—my own views have undergone a change. Herein I have been much impressed by the distinction which our German colleagues have drawn between "*wirtschaftliche Heilung und wissenschaftliche Heilung*"—as we may express it in English, between a pathological cure and an economical cure. What is meant by a pathological cure we know, as we know also that the means of such cures are unattainable save by persons of wealth and leisure, and not by all even of these. Keeping our minds fixed upon the first stage—for we will not admit that physicians will continue to let cases drift into the second without sanatorium treatment—it appears that after a certain time patients may be allowed to return to clean homes and wholesome occupations so far improved, and, what is quite as important, so deeply imbued with sanatorium methods, that most of them will hold their ground for some years, and many will ultimately recover. To fix a term for the individual case is, of course, impossible; but, now that large numbers have been dealt with, a mean term may fairly be demanded of us by the paymasters of these resorts. Some sanatoriums put the mean term at three months, which seems to me too sanguine; others put it at six months.

When we try to apply some general rule to the individual case, to estimate for the patient and his friends how far he will fall within the mean or without it, we must be guided by certain individual features. For instance, if fever continue after the first ten or fourteen days, the mean duration of residence will be exceeded. How long may fever continue, and yet the patient do well? In view even of an extreme term, is it any use keeping a patient in the occupation of a bed if the fever has not subsided, say, in four or six months?—for we may have six or eight per cent. of such cases. Rapidity and instability of pulse are adverse symptoms. Sugar or albumin in the

Digitized by Google

urine is, of course, of bad omen. Poor feeders do badly; topers do badly. Young patients not out of their teens do badly on the whole; and so, again, do patients in the later decades of life. A steady gain of weight is, of course, hopeful, but patients may gain weight while retrogressing in respect of local disease. I have expressed the opinion that tuberculous pedigree does not tell against the case prognosis; but herein Sir William Broadbent differs from me. Pregnancy is, of course, a very unfavourable complication. Gout I regard as a very favourable one. An obedient patient of tranquil temperament has a much better chance than the wayward or fretful. As to physical signs—I have not worked in a sanatorium; but, on my fragmentary experience, I think that, if on admission there were crepitations at one apex, these ought to have dried up in a month or six weeks if the patient is to be discharged in three months, at which term the physical signs should have become fairly negative. As to bacilli, these pests seem to persist indefinitely in many patients discharged and otherwise doing well. In bronchiectatic cases, on the other hand, they may not be found. I attach more importance to a diminution of the quantity of the sputum. Upon such data as these I think the chances of the individual case in respect of the mean stay may be calculated, say, after the first month's residence. That the presence of tubercle in other organs is a very grave factor in the prognosis I need not say. Cases of slow scrofula with pulmonary complication often, indeed, do remarkably well, but in most other cases of multiple diseased organs I have had disastrous experience. Multiple deposits in the lungs themselves also in their degree forbid the more sanguine hopes. However, if sometimes we are disappointed by the ill results of a case in which we had looked for better things, on the other hand it happens, more frequently, I think, that cases in which we had not dared to hope much gratify us by revealing an unexpected power of recovery. Thus the cruel face of Nature may break into a smile.

It is only by means of the kind reception that many sanatorium physicians have given me, and of the free and generous manner in which they have allowed me to share their thoughts, that I shall dare to-day to discuss treatment in sanatoriums. I will begin by quoting the words of him to whom my debt is the greatest, viz., Henry Bennet. In 1866 he wrote of the physicians of his time: "They dare not apply to their patients the ordinary rules of hygiene; they dare not give plenty of animal food; they dare not give fresh cool air day and night; they dare not keep the skin clean and cool by cold or tepid sponging. . . . Their patients, when they go to health resorts, think they ought to take exercise, and they do so. . . . They think it did them good when well, and will do so now when they are ill; but they merely walk themselves into the grave." If I quote no more than this, does not that which I have read tell us how enlightened a forerunner was Bennet in the treatment of phthisis? And now we are tending to drop into the opposite routine of idleness and stuffing. So hard is discrimination. Whatever our treatment, let us first take such order with ourselves that it shall not be treatment of the abstraction Phthisis, but of the individual victim of tubercle bacilli, of which

Digitized by Google

victims no two are identical. Such discrimination means well-paid physicians, and many of them. I repeat, let us not fall into the routine of asylums for the insane, wherein the public authority has always been seeking how far it can reduce the salary for which it can get medical men to apply, and, having got them cheaply, how many patients it can make them attend to. Let not the public act in sanatoriums as it acts in asylums and hospitals—as if, that is, the public had itself found out how to cure diseases, and had engaged a few medical men to carry out its own ideas.

As in other diseases, so in phthisis the first task in undertaking a cure is to clear up arrears. When the patient comes under care, in most cases, at any rate, he has drifted into physiological debt. He has mortgaged his estate, has overdrawn his banking account, has bought raw material at forced prices and paid for it with bills renewable at heavy fines. Our first duty, then, is to straighten the account; and it is astonishing how much is to be done by reducing expenditure, by husbanding profits, and by clearing off arrears. Every patient, febrile or not, should rest for ten days or so, until the physician can reckon up the balances. And let it not be forgotten that the stomach, indispensable as it is, indeed, because of its indispensability, needs rest too—that is to say, it must be nursed. To thrust a heap of food into the stomach of a worn or exhausted human machine is to court failure; fortunately, such is the tolerance of the human body, the patient is often quit of such an error for ten days' indigestion and "biliousness," when the food has to be moderated after all. Now I do not often read in the reports of cases, amid their long lists of physical examinations, records of the dimensions of the stomach before and after meals, which I regard as one of the most important of these examinations. In weak and febrile patients the walls of the stomach are almost always lax, so that the food is delayed in the viscus, and tends to ferment there, especially if the secretions of the peptic glands are attenuated. Under the internal pressure the organ gives way more and more, and may not recover itself in the hours of emptiness, or, indeed, if feeding by frequent snacks is advised, may never become empty. Not long ago I saw such a patient, stuffed on the most modern principles; his medical attendant was annoyed that he positively declined to eat, that he was nauseated, and was losing ground rapidly. On examination of the emaciated body, we were able not only to map out, but even to grasp, a large pendant stomach as full of food as a haggis. How long it had been full I do not know. For the first few days let rest be rest all round, stomach included; then, after the varying dimensions of the organ are known, the food may be increased according to the energy of the individual. Another warning I would give is to watch carbo-hydrates with care. Feeble stomachs digest these materials with less activity, and they tend to generate flatulence. Man at bottom is carnivorous. I think milk is given too profusely. The bulk of liquid has two dangers—first, it impedes digestion by diluting the gastric juice and distending the stomach—at any rate, in non-febrile cases, where liquid is not so quickly disposed of; and, secondly, it adds to the mass of the blood which the heart, often ill-nourished, has to lift. For my part, I rarely find the need of forced feeding. Now and

Digitized by Google

then a patient is fastidious; but the secret of forcing the food is to put the patient into open air as cool as he can bear it, and, if the cook is a good one, the appetite in hopeful cases will come back of itself. On my first visit to Davos, when I found myself at table with thirty weather-beaten people eating like wolves, I exclaimed to Ruedi, "But where are your patients?"

Food has two ends to fulfil—to compensate fever, if, that is, fever be present, and to nourish the body. Feverish patients will dispose of more liquid than the non-febrile; and as in them the stomach is usually relaxed, the food must be in frequent small quantities. Gabrilowitch, in a series of careful experiments, has shown that in the phthisical, and not in the febrile only, the weight falls with surprising rapidity between meals if these are at too long intervals. This fall he finds best marked at nights, between supper and breakfast. It would seem, then, that so long a fast, however wholesome for the sound, is not desirable for the sick. As he epigrammatically puts it, "Irritability and sleeplessness are the hunger of the phthisical." When fever has disappeared, and the patient has regained his normal weight, the physician, by close watching of the scales, will be able to return to ordinary meals at the usual intervals. There are now no more arrears to make up, only the balance of waste to repair.

Of alcohol Bennet well said, "Even when it is prescribed medicinally, there is always the risk of abuse. It is a double-edged sword." For a few days, occasionally, when the patient flags, when his skin is cool and damp, when his pulse is feeble, when his digestion is slow, and when after meals he is depressed in spirits, it is helpful; as a rule, it is not needed. It is our duty, then, as pathologists, knowing that liquorish habits favour tuberculosis, to discourage the still prevalent notion that without alcohol a sick person can never regain strength. There are many lives to throttle out of this cat yet. All patients, without exception, must rest both before and after meals. The latter they remember; the former, which is at least as important, they are apt to forget. Patients liable to vomit food must take their meals in bed. Exercise may be insufficient; indolence may lead to padding with fat rather than to hard condition. Stalled fat cattle are more subject to tuberculosis than the leaner kine on the hillside. The restoration of the heart degenerated by toxins is to be brought about by regulated exercise, and in no part of the treatment is the discrimination of the sanatorium physician more indispensable. On massage I would invite opinions. In former years, when more patients remained under home treatment, I thought it very beneficial; since the departure of our patients for the sanatorium, I have had little further experience of it. I would ask particularly whether massage is inadmissible during moderate fever?

In the earlier days of Davos, hydropathy had some vogue, especially the douche. On these means also I would invite discussion. I have found much good in the wet sheet, and the wet pack may be helpful in fever.

The use of gymnastics in the treatment of phthisis is little understood. That in certain phases of disease of the lungs gymnastics must be inappropriate needs no insistence. For instance, he would be a brave man who ordered

Digitized by Google

lung gymnastics in a case of softening or recent hæmorrhage. In phases of softening, again, the risk of suction of septic matters from one part of a lung into another would forbid all exertion likely to cause forcible inspiration. At high altitudes it would seem that the larger volume of the inspirations promotes insensibly an expansion of the lungs too gradual to bring about this peril. In healing stages, when softening has ceased, and the lung is drying and laying down protective fibre, may not gymnastics, under supervision as skilled as for cardiac disease, do much to expand, and thus to call into healthy function, the parts which the tubercle has spared? I seek the answer from those who are dealing daily with these problems.

To admit that there is no drug endowed with specific virtue in phthisis is not to admit that drugs are never useful. For the most part, the open air will reduce fever, will prevent sweats—whether due to fever or debility—will promote appetite, and will calm and fortify the circulation. But it is not to be forgotten that during liberal feeding gentle laxatives, and especially an occasional mild mercurial alterative, prove valuable; that a drop of an arsenical solution may settle an irritable stomach, a few drops of strychnine may brace up a slack one, and so on; but I have no faith in courses of drugs, whether antidotal, tonic, or topical, save, of course, in laryngeal cases, which, as the part can be got at, are to be treated locally from the first.

Finally, I must protest against the emptiness of mind which certain reformers would enforce upon their patients. I feel sure that a lack of tranquil occupations and amusements conduces to introspection, and, moreover, is not without grave peril to the moral life. Most of the patients we have to treat depend for their livelihood upon habits of industry; young folks may be sent to a sanatorium at the very time when the habits of life are forming, and it is deeply to be regretted if to the calamity of tuberculous infection must be added a dissipation of those virtues of energy and method upon which our happiness depends. During phases of bodily incapacity submission to the inevitable takes the form of a duty, unfortunate as the loss of time may be; but vapid hours and aimless days may break up the hardly won discipline of a careful education, if it do not abandon the thoughts to wantonness. The vacuous looks and aimless wanderings of the patients hanging about the precincts of some sanatoriums have impressed me painfully. Vigorous games are rarely suitable; but surely there are occupations, such as gardening, the fine arts, literature, natural history, and quiet handicrafts, which to those free from fever would be beneficial. Perhaps even the visits of teachers in the arts and sciences would not prove so wildly exciting as to throw the population into a fever.

I will sum up the chief questions which I have propounded.

1. Can mixed infections be recognised from fever curves?

2. Can we distinguish between economical (*wirtschaftliche*) healing and complete (*wissenschaftliche*) healing? if so, what is the mean term of residence for the economical healing of early cases?

3. How long in certain active cases, say, from 6 to 8 per cent., is a febrile patient to be kept to bed in the reasonable hope of recovery? For instance,

Digitized by Google

in a public sanatorium are we justified in retaining patients who have been confined to bed for six months, five months, or even for four months?

4. What estimates of improvement, and what rules of prognosis can be based upon physical signs alone?

5. Is multiple tuberculosis, for instance, in lung and testicle too hopeless a condition for a public sanatorium? How far is it comparable with an equal extent of mischief in one organ?

6. Of what use, if any, is massage?

7. Of what use, if any, is hydrotherapy?

8. Are special pulmonary exercises appropriate at certain stages of progress? and if so, when, and under what conditions?

9. Must we repair the body at the expense of the life of the mind? Can we not give even some educational value to the sanatorium besides the medical drill of it?

II.—By J. KINGSTON FOWLER, M.A., M.D. Cantab., F.R.C.P. Lond.,

Physician to the Middlesex Hospital and to the Brompton Hospital for Consumption, London.

IN November, 1899, I had the honour to open a discussion on this subject before the Royal Medical and Chirurgical Society, London, and it is not necessary for me to go over again all that I said then. At that time it required a little more courage than it does now to stand forward and speak in favour of sanatorium treatment. I have little to retract from what I said then. Subsequent experience has only confirmed me in the high opinion I had formed of this method. I think that whatever may be in store in the future with regard to the therapeutics of tuberculosis, a system which has for its object the development of all the powers of the body, and the increase of the resisting power of the individual, must always hold a place. I spoke in the discussion that I have just referred to in praise of Brehmer, and, as Professor Allbutt has said, I do not think anything has been added of value to what Brehmer laid down as to the main principles to be applied in the treatment of tuberculosis.

I am glad to find that in Germany it is proposed to raise a monument to Brehmer. Those in this country who value his work will do well to subscribe to that monument.

I also spoke strongly in favour of the system of treatment which Dr. Walther had developed at Nordrach. I am glad to know that Dr. Walther's system is fully appreciated in this country. Perhaps in some particulars he may have gone to an excess, but I would say that a man of his enthusiasm is able to impress his patients and the public, perhaps, to a greater extent than a man working on a more scientific method, although I should be sorry to say that Walther's method is not scientific.

Digitized by Google

I suggested that the name to be applied to this treatment should be "the sanatorium treatment." I am glad to welcome Dr. Allbutt in support of that. It is in a sanatorium that this method can be most completely carried out. I constantly see patients whose friends speak to me of the southern aspect of their house, of their beautiful garden, its high, dry situation and its suitable soil; that spells disaster, in the greater number of cases, if you give way to it—at least that is my experience. There are persons, no doubt, who have great self-control, and who can be trusted under these circumstances to the care of a physician who is versed in the method. But for the great majority of patients it is very much easier and very much better that they should go into a sanatorium, even if only for a short time, for there they learn how subsequently to conduct their lives, and, so far as I have observed, they do not readily afterwards depart from it. They can be trusted afterwards, they can scarcely be trusted before.

The aim of treatment should be to secure, as I said in my address, that the lesions are obsolete—that they will stand the stress of work and life. The cure of the patient, the education of the patient, and the facilities for research, are what we look for in sanatoria. I said then that there was no reason to suppose that this method could not be carried out equally well in this country as it is in Germany and elsewhere. I think the experience we have had has shown that that forecast was true.

Into the details of the sanatorium method I do not propose to enter now. I would only make one remark, with regard to the question of feeding: that weight may be gained, and that the disease may not be undergoing arrest is now obvious. I think what we require is careful scientific work on metabolism, which will enable us to determine the amount of food which the patient can retain with advantage. I am glad to find that my friend Dr. Bardswell, who began his observations at the Brompton Hospital, is continuing them, and has received a grant from the Royal Society for the purpose. We shall in time obtain data which will be of the greatest possible use to us in determining the amount of food which is desirable in any given case.

In the paper which I published in 1892, on "Arrested Pulmonary Tuberculosis," I gave the results obtained in the post-mortem room of the Middlesex Hospital from 1879 to 1886, where, in 1,943 autopsies, I had observed 177 cases, or 9 per cent., in which there was arrested tuberculosis. The percentage has been placed very much higher by other observers. Dr. Sidney Martin, who continued the observation, brought it out at almost exactly the same figure. In 134 of these cases the disease was arrested in its early stage. In 43 cases it was stated that a cavity was present. I do not think it follows that a cavity was present in life in those cases. We may learn from Nature—and as Nature arrests the disease far more often in the early stage, it is in that early stage we should endeavour to obtain the cases.

It was stated at that time that these were not cases of phthisis; they are cases of pulmonary tuberculosis, and I suggested that that was the proper name for the disease, for it is surely unscientific to give a disease one name in the early stage, and another name in the later stage. If you leave the cases

Digitized by Google

long enough, they will be cases of phthisis. I think we should do better to speak now of "Pulmonary Tuberculosis."

The interesting paper which Dr. Burton-Fanning is about to bring before the Congress shows that in 88 per cent. of the cases only one lobe was affected, and there were no signs of cavity. In 88·4 relative recovery was obtained, which shows quite clearly that in the early stage the best result is to be looked for. I have been in the habit of endeavouring, as the result of an examination, to form a mental picture of the condition of each lobe of the lungs. If you conduct an examination, and are prepared or obliged to write down at the end of it exactly what you think is the condition of each lobe of the lung, you will make your examination with much greater care than if you are going to put down "Ph. II." or "Ph. III."

There are other facts to be taken into consideration. But, as a general rule, we shall find that according to the extent of the disease will be the excellence of our results: that where one lobe is affected, or perhaps the upper lobe on one side, and the lower lobe on the same side, we shall get the best results.

I will say one word with regard to consumption hospitals in towns, and the use that they can still be. It is quite clear that sanatoria in the country are the best places to treat the disease, but still hospitals in towns may be of great service. In the first place, they are centres to which patients go, and there the patients can be educated. We should make it a condition that no patient should be treated there who would not undertake to so manage his expectoration as to be of no possible risk to others. If that point is insisted upon, much good will be done. There should be places from which patients can be drafted to sanatoria in the country. We should endeavour to get the subscribers and governors to give up their right of admitting patients to the wards after the patients' names had been on the register for some time. The governors should be urged to give up their in-patients' letters altogether, and to give to the physicians the opportunity of filling the beds with those cases in the early stage of the disease in which alone treatment can be successful. I feel sure that all who have worked at a hospital for consumption must have seen hundreds of cases which after six weeks or two months of waiting had passed the stage in which recovery was possible.

I have been very much struck to find in a town like London how readily patients become accustomed to a completely open-air life. At the Brompton Hospital we have several wards set apart for this method of treatment. I have constantly asked the patients who were in places where the winds of heaven were blowing freely, how long it took to get accustomed to the life, and they usually replied "two or three days."

It is certainly remarkable that patients amongst the poorer classes should in three days be able to live a completely open-air life. I was assured by the house physicians and the sisters of the wards, and the patients themselves, that patients in wards with the windows open and fires burning suffered from the irritating effects of fog to a far less extent than those in other parts of the hospital.

Digitized by Google

With regard to sanatoria for the poor, Dr. Allbutt has pointed out how long a period is necessary to obtain complete obsolescence of the lesions. I think we shall find that it is only those who have no one dependent upon them, the unmarried of both sexes, who will be suitable patients for such sanatoria. It is almost certain that if you take men who have children who are dependent upon them, the wives will come down and say, "You are looking well—we are starving; when are you coming back to work?" We shall never, under these circumstances, induce them to stay long enough to produce arrest of the lesions. My friend Dr. Collier, of Oxford, writes in the *British Medical Journal* of this week: "Some years ago we started a Ventnor Fund connected with the Radcliffe Infirmary at Oxford, and retained a small number of beds at the Ventnor Consumption Hospital, to which we sent our most promising cases of early phthisis; and the results were for the time most instructive and satisfactory. Patients after a few months at Ventnor would return to Oxford different beings. All their symptoms had abated, and often the resident medical officer at Ventnor would report them as cases in which the disease was arrested. For a few years every Christmas I wrote to the friends of patients who had been at Ventnor a year before to inquire about them, and I invariably found that within a few weeks or months of their return to their old surroundings, the disease had broken out again, and I was never able to find evidence of a single case cured or permanently arrested; in a longer or shorter time they all died."

I spoke to Dr. Walther some time ago, and he gave me an experience which was exactly similar. There were a certain number of beds at his sanatorium, maintained by a Philanthropic Society in Germany, who sent some working men there. They improved to a great extent whilst under treatment. They returned to their work, and in a very short time they all relapsed, and it is probable—he assured me he thought so—that they lived a shorter time than they would have done if they had never been submitted to sanatorium treatment at all.

We must endeavour to enlist in the service of the consumptives, philanthropic individuals, such as those who are now giving their attention to epileptics. I read in the paper last week that the David Lewis Trust, of Liverpool, had purchased an estate of 500 acres, of which 130 was appropriated for a colony for epileptics. We must endeavour to bring before the millionaires of this country, or America, or both, the advantages which would accrue to the consumptives if they could only be provided, after they have obtained in sanatoria obsolescence of their disease, with opportunities for working on the land. I think if we could induce them to start horticultural gardens that we should do a great work, and I hope that one result of this Congress will be to elicit on behalf of the consumptive the sympathies of the wealthy of this country.

Digitized by Google

III.—By R. W. PHILIP, M.A., M.D., F.R.C.P. Edin.,

Physician to the Victoria Hospital for Consumption, Edinburgh.

In the remarks which follow, I shall endeavour to refrain from the expression of merely academic opinion regarding the effect of what has been termed the sanatorium treatment of pulmonary tuberculosis.

I prefer on the present occasion to base my statement on personal observations which have been continued for some ten years. These have concerned a large series of patients. The list includes more particularly some 700 patients treated on more formal sanatorium *régime* under my eye at the Victoria Hospital for Consumption, and at Woodburn Sanatorium, Edinburgh. It includes also patients who have been subjected, on my advice, to similar treatment at other sanatoria or privately. While I am familiar with the methods of, and have visited, many sanatoria both in the United Kingdom and abroad, it seems desirable to restrict the present survey to "facts observed by myself."

THE SYSTEM.

It may be convenient if I outline in a word or two the leading features of the system as I conceive it.

In the first place, while the principles of the system are physiological and correspondingly definite, there can be no hard-and-fast rule applicable to every case. The detailed application varies in different instances. Herein lies one of the dangers both to the system and to patients who happen to be committed for treatment into the hands of persons who regard the matter as largely rule of thumb, or swear blindly by the precepts of one master. Each case requires consideration *per se*. At the same time, certain broad lines are followed. These may be considered under the following heads:—

1. *Open Air.*—There is no doubt, notwithstanding much that has been written in qualification of the statement, that hyperaeration (as conventionally we are wont to conceive it) is the major agent in the treatment. It is the one essential—the *conditio sine quâ non*. While difference of opinion as to dietary, as to the relative amount of rest or activity, may be admitted in different cases, the uninterrupted exposure of the patient to the influence of fresh air is applicable in every instance. In my experience there cannot be too much of this.

I remember in the early days feeling a certain timidity as to the length and degree of exposure of the patient. We were careful to attain a fairly constant temperature in dwelling- and bed-rooms occupied by patients. It used to be an instruction to our nurses that the temperature should be regulated by the thermometer placed in each room. Especially were more delicate patients thus carefully handled.

Digitized by Google

Experience has modified this greatly. I allow the freest access of fresh air to the patient day and night, at all seasons, provided he is sufficiently clad. I am not careful as to the temperature of the surrounding air, whether in or out of doors. Indeed, there seems a disadvantage in endeavouring to combine free exposure of the patient out of doors with his occupancy of rooms whose temperature is maintained constant, or, still worse, which may become overheated. It is more logical, and experimentally more successful, to subject the patient indoors to approximately the same conditions as out of doors. Assuredly let him be comfortably warm by means of clothing, and, if necessary, by hot bottles, but let us be sparing of artificial heating of the atmosphere.

It is an interesting commentary on this statement to add that the results during the series of years I have mentioned have proved themselves in many respects more satisfactory in winter than in summer. Even with the most delicate patient I have on no single occasion observed a bad result follow the most complete exposure to such influence. Contrariwise, in proportion as the patient is more freely exposed the satisfactory result is attained more quickly. During the present spring, for example, I have allowed a considerable number of patients not only to be out all day but to sleep outside all night, merely under cover of a three-feet-deep shelter, which is completely open to the front. Some of these patients literally ran ahead as compared with other patients sleeping indoors, and in the entire group the results were uniformly satisfactory. Subject to reasonable care in respect of clothing, there cannot, in my experience, be too free exposure to the open air.

2. *Rest and release from disturbing conditions.*—It is a mistake to speak as if the system of rest were one, and the system of exercise for such patients were another, thing. Neither the Liegehalle nor hill climbing is applicable to every case. These should not be pitted against each other as if they constituted opposing modes of treatment.

For almost all patients rest is the immediate indication when treatment is commenced. This must be maintained for most patients as part treatment, at least for a long time. The determinant of the necessary degree of rest is especially the state of the pulse and the temperature. It is desirable that the patient should rest chiefly if the temperature oscillate from day to day above 100·5 Fahr., or if the pulse rate be maintained above 90 per minute.

The negation of rest means in such cases greater irregularity of temperature and increase of pulse rate, with other evidence of circulatory prejudice. It is remarkable how certainly an apparently slight error in this matter registers itself in elevation of temperature and circulatory disturbance. Even the visits of friends, the receipt of letters, or similar incidents may exercise a definitely harmful influence.

3. *Exercise.*—As improvement proceeds, as evidenced by the general condition, and more particularly by the pulse and temperature, the necessity for complete rest is removed. The patient is allowed to take short turns, measured by minutes at first, and then of greater extent, once or twice in the

Digitized by Google

day. Such exercise is begun on the level and gradually allowed on an incline, until even hill climbing becomes possible. At all times the rate of progression should be slow, and care be taken that the respiratory movements are free and full.

As the necessity for rest lessens, the intervals of activity are correspondingly lengthened. The effect of activity should be gauged by careful record of pulse and temperature. At this stage, too, simple respiratory exercises often prove of service. Many patients have much to learn regarding the physiology of respiration.

4. *Dietary.*—Alimentary disturbances form leading features of the disease. These are corrected by a carefully studied dietary, of which the patient must be induced to take a sufficient quantity. This is not the same as "forced feeding," at least not in the sense in which that term is sometimes realised.

The dietary of each patient, and of the same patient at different periods of his illness, is worthy of most careful consideration. There is reason to fear that frequently enough an unsuitable dietary is prescribed, a crude attempt is made to ram the material into the alimentary tract, and the patient perhaps rudely handled if either he or his stomach rebel.

On the other hand, if physiological conditions are fulfilled, the patient generally eats of his own accord. He frequently becomes voracious. In order to reach this end, he must rest sufficiently before and after meals and the bowels must be regulated with care. The rules regarding fresh air day and night must be followed strictly. It is often of much advantage that the patient takes meals in the open air.

It is impossible, in the limit of time, to enter further on details of dietary.

5. *Skin hygiene.*—The condition of the skin should receive attention. This is not merely a question of cleanliness. From the point of view of heat regulation and excretion, healthy skin activity is demanded. Hence both clothing and bathing should be carefully considered.

The clothing should be sufficiently warm, but preferably of light weight and porous. Shetland wool materials afford an excellent example of the sort of thing that is wanted. The absurd overclothing which was used formerly, and indeed is still sometimes sanctioned, is to be condemned. Chest protectors of all kinds are wrong. The patient must get rid of the notion that he got his disease by exposure and that he will cure it by protection.

The regular use of a rapid cold bath is in most cases not only possible but most beneficial. Sometimes improvement in a patient's state commences only when cold bathing is undertaken. The temperature of the water is of course to be regulated according to the season, so as to avoid undue shock. Care should be taken that the patient is quickly and comfortably dried after the bath, without over effort to himself.

6. *Medication.*—It would be out of place at this time to enter on the discussion of the value of drugs in the treatment of the disease. It is remarkable how comparatively few drugs seem necessary when the more cogent physiological indications have been fulfilled. None the less is it true that the significance of medication becomes greater if the use of drugs be

Digitized by Google

directed from day to day under careful surveillance. Particularly does this hold good in respect of tuberculin, which has proved itself in a considerable number of my cases of immense diagnostic and therapeutic service.

The Site of the Sanatorium.

My experience has led me to the opinion that a sanatorium can be suitably established, with the expectation of excellent results, practically anywhere, provided that the immediate surroundings be free and open and there be a sufficiency of ground for the carrying out of open-air methods, and directly noxious influences be absent.

It is of course inadvisable that the sanatorium be actually within our large cities. On the other hand, it is not necessary, nor indeed is it from several points of view desirable, that the sanatorium be too far removed from the town or district in which the patient usually lives and has to make his livelihood.

If it is possible, as my experience indicates it is, to obtain cures under the normal climatic conditions of the individual, this is most assuredly to be preferred. For thereby the patient, and the wider circle of persons whom the patient influences, are made to recognise that the prevention and cure of the disease lie within their own power, that is, are possible at their own door. The two sanatoria in which my observations have especially been made are both of them situated within three miles of the centre of Edinburgh, and I am satisfied, both by direct observation and comparison, that the results have been no less satisfactory than the best results elsewhere.

Results.

In the foreground it may be stated without fear of contradiction that the results obtained on sanatorium *régime*, whether actually at a sanatorium or otherwise, are much in advance of the results which used to obtain on the more or less protective system. Indeed, I am bound to say that the results have been uniformly good when the condition has been taken in hand reasonably early.

In no case have I seen harm accrue. The results of treatment, even in the worst and most advanced cases, are such as seem to justify the mode of treatment no less than do the effects obtained in early cases. It is true that dying patients are not recreated by the system. But the change in the type of disease which occurs even in such cases when physiological treatment is adopted, has seemed to me one of the most remarkable facts of medicine. The speedy disappearance of the more distressing symptoms, and the return of comparative comfort, are striking. Even if death be the final issue, much has been done towards a euthanasia.

It is, however, in the recovery of incipient and comparatively early cases

Digitized by Google

that the curative significance of the method is especially witnessed. In these the results which have come under my observation enforce the view that, given the opportunity for a sufficiently long and satisfactory course of treatment, the result will be almost certainly good.

It may be serviceable to individualise some of the more evident results :—

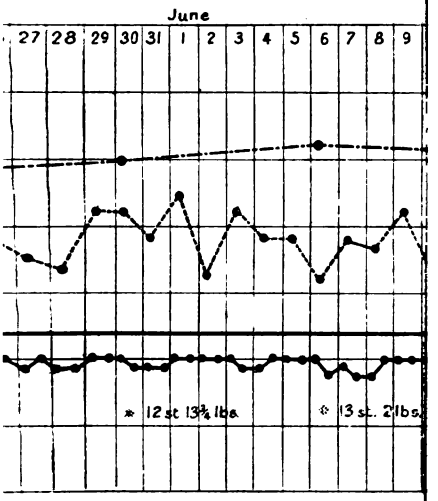
1. *General Condition of the Patient.*—The aspect of the patient is very different from that of the patient treated on protective lines. It is amazing how quickly the change is effected, and the worn, discoloured look is replaced by the appearance of ruddy health. Many members of the Congress must be familiar with the picture. For those who are not it is difficult to describe the change in words without seeming to exaggerate. It seems simpler to ask you to look at the photographic representations of patients which I have placed in your hands. I would ask your especial regard to one, that of a gentleman who had for many months sought for cure of grave pulmonary and laryngeal disease by residence in reputed climatic resorts, and who at the end of two years' time was in the condition indicated on the left-hand side. The portrait to the right represents the appearance of the same patient after ten to twelve months' sanatorium treatment at Edinburgh. I do not think I exaggerate when I say he looks another man. The others are hardly less striking, and were there space for freer illustration, I might multiply this abundantly.

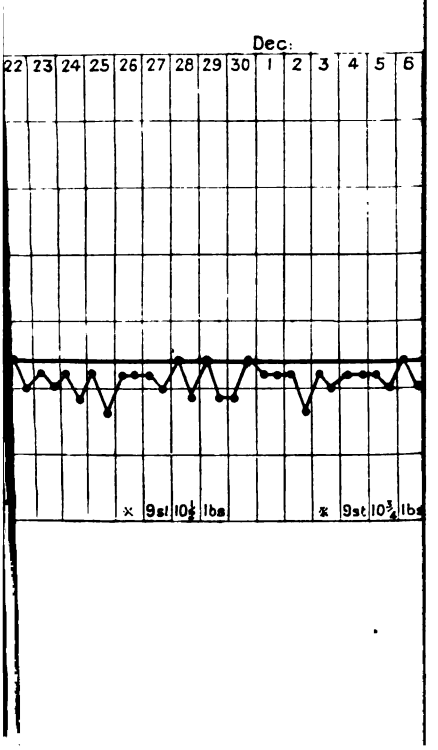
Nor is the change confined to appearance merely. Flesh is rapidly made. Thus, for example, patient No. 2 gained within six months some four stone in weight. A gain of from 2 to 4 lbs. a week is common, and 8 lbs. or more have been registered in my experience. More important than mere gain in weight, the flesh which is made is good solid stuff when the dietetic programme is satisfactory. The muscles, no less than the adipose tissue, increase in bulk and become plump and firm. Myotatic irritability and other evidence of want of muscular tone tend quickly to disappear.

2. *Temperature.*—The effects on temperature are highly noteworthy. It is matter of everyday experience to observe that swinging temperatures—oscillating perhaps from sub-normal to 101° F. or thereabouts—are gradually modified so as to approach the normal.

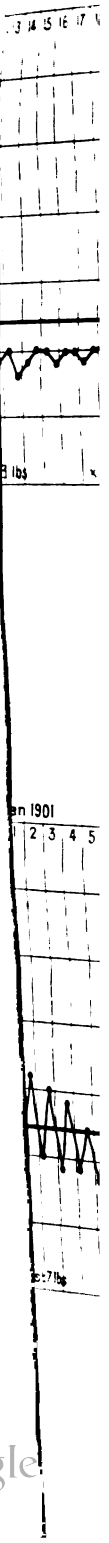
In going round a properly managed sanatorium it is exceptional to find much evidence of pyrexic disturbance among patients who have been any length of time in residence. This is not because the physical condition of the lung is satisfactory. Contrariwise, it is the commonest thing in the world to find abundant evidence of moisture and vomica formation in the chests of persons whose temperature, taken four-hourly or even every hour, is strictly normal. Here, again, the fact is best illustrated by actual reproductions from the charts of patients, and I place in your hands several such records carefully reproduced for me by Drs. Mears and Galbraith, of Edinburgh (Charts 1–4). In all these the return of the temperature to the normal after prolonged periods of grave pyrexial perturbation is remarkable

Digitized by Google

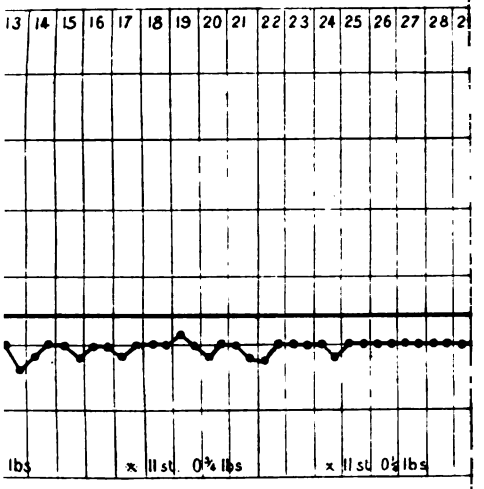


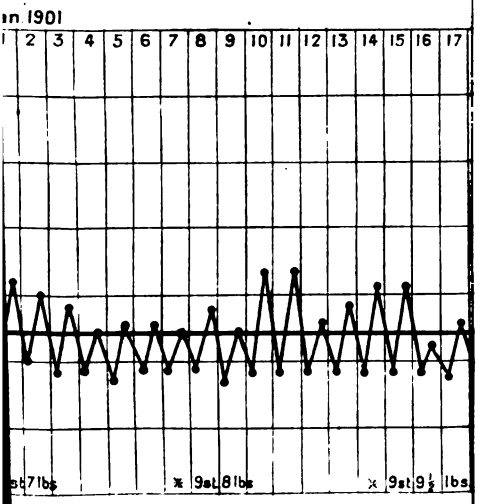


Digitized by Google



Digitized by Google





Digitized by Google

Digitized by Google

The uninterrupted uniformity of temperature thereafter for months is most striking. Such illustrations might be repeated indefinitely, and commonly the result is attained apart from special drug treatment.

3. *Circulation.*—There are few more beautiful phenomena in clinical medicine than the change effected on the rapid, soft, and wavering pulse of the tuberculous patient when for the first time he is placed on the physiological line of treatment.

Within a day or two in most cases, and with few exceptions within a week or two, the rapid soft pulse is replaced by a slowed and comparatively resistant beat. I regard the less or greater degree to which this is established as immediately of great prognostic value. Chart No. 1, which I place in your hands, shows in the dotted line the alteration in rate of pulse coincident with the fall of temperature (black) and increase of weight (dots and dashes). The tendency to coldness of extremities and shiveriness similarly goes.

4. *Alimentary Disturbances.*—Anorexia, digestive discomfort, and other alimentary disturbances, which form distressing features in most cases under the usual system of treatment, are reduced to a minimum.

I have already referred to the return of appetite. The disappearance of the dyspeptic phenomena is no less remarkable. Measures specially directed to the stomach commonly become unnecessary.

5. *Night Sweats.*—In nothing is the change of type produced by the physiological line of treatment rendered more evident than as regards night sweats.

This classic symptom disappears entirely. A patient who may have perspired so much as to require a change of nightdress three or four times a night loses the tendency within a few days, or at most a week or two. So true is this in my experience that I am in the habit of teaching students to regard night sweating as largely an expression of ignorance or culpability on the part of those in charge. I can hardly recall the time when last I ordered special drugs for the symptom.

6. *Other Symptoms.*—Cough, expectoration, and dyspnœa lessen similarly. There is, in fact, little cough to be heard, save among the new-comers.

Of special interest is the comparative infrequency of hæmoptysis. Apart from faint streaking of expectoration with blood, I can almost count on my fingers the occurrence of hæmoptysis among the cases included in the present review. Yet the list includes many cases of pronounced and advanced disease.

It is needless to enter here on the question of physical signs, which improve correspondingly.

General Conclusions.

In this rapid survey of so large a subject it has been possible only to accentuate certain of the more salient points. It may be frankly yielded that these are sufficiently striking to warrant the belief that in the system—by

Digitized by Google

whatever name we choose to call it—there lies much hope in relation to tuberculosis.

The conception of pulmonary tuberculosis in the past has been, and still is by many, too much that of a localised lung disease, while the constitutional condition has been insufficiently considered. So far as treatment is concerned, the reverse must be the case. The constitutional rather than the local disorder must be kept in view.

The systemic disturbance is immense. To combat this successfully implies a vast systemic reformation. A return to the physiological life must be the aim of treatment, whether conducted in a sanatorium or at home.

The observed phenomena indicating the tendency towards *natural* cure are in no disease more striking than in tuberculosis. The chief aim of treatment must be to limit as much as possible those factors which diminish the normal resistance of the tissues, and to interpose no obstacles in Nature's path through clumsy attempts to hasten cure.

I believe the open-air or sanatorium treatment has come to stay, because the method is physiological. Not even when the therapeutic activity of tuberculin is rendered more definite can a method which is no less prophylactic than curative be discounted.

It has been asked, Can the system be carried out elsewhere than in a sanatorium? Can the method be carried out at home? My answer is that it may be practised at home, that it may be practised anywhere, provided there is sufficient accommodation, a sufficient appreciation of what is necessary, and a sufficient determination to carry this out at all cost.

It is not easy to realise these in practice. In order to do this it is desirable that the patient, the nurse, and doctor have had some experience of sanatorium methods. The educational value of sanatorium life can hardly be exaggerated in relation to the patient. Hence in some cases, where it is impossible to extend the patient's residence at a sanatorium to the full limit, it may be worth while to sanction his residence there for, say, a couple of months—and this from the educational point of view—so that faulty home conditions may be amended on physiological lines with the intelligent acquiescence of the patient.

A further grave difficulty in the way of home treatment exists commonly in respect of the patient's relatives and friends, who, in the name of kindness, or on the plea of common-sense, are apt to come between the patient and his cure. It may reasonably be expected, however, that this difficulty will tend to disappear in time, and the physiological *régime* become more generally applicable. There is little doubt that already a considerable wave of education has passed over the country through the labours of individual physicians, the measures which have been adopted by public health departments, and the efforts of the National Association for the Prevention of Consumption. It is to be hoped that in this respect good fruit will result from the session of this Congress.

The measure of success in treatment will depend on the correctness with

Digitized by Google

which the physiological indications are fulfilled. Much must therefore depend on the physician, who is chiefly responsible for the *régime* and surveillance of the patient. Much will depend, too, on the earliness or otherwise of diagnosis. The comparative certainty of cure in early stages of the disease affords one of the strongest incentives to exactness in early diagnosis. A week or month lost in relation to the determination of this may require to be multiplied many times in relation to the treatment of the disease.

Further, much patience will be required, both on the part of the sick man and his attendants, in order to prevent disappointment as to the final result through undue curtailment of the period of more rigid treatment. No less disappointing may prove the later attempt to force Nature to maintain a cure which has been achieved with difficulty under physiological conditions, under conditions the reverse of these.

Hence in all cases, but more especially among the working classes, the question of after-life becomes one of pressing moment. It is disappointing to find that patients who recovered perfectly well, and who retained recovered ground for months or longer—indeed, so long as they were able to lead a physiological life—have gone back to the old state, or still further, simply because, by force of habit or necessity, they fell from the physiological path.

It was in view of the tendency to such declensions that some years ago I ventured to point out the great desirability which exists for the establishment of some system of *tuberculous colony* in which the convalescent and convalescing patient might each contribute by work, according to his physical capacity, towards the common good, remaining the while for his own benefit under suitable medical superintendence.

When our municipalities and local authorities seriously tackle the treatment of early cases this aspect of the question should be kept in view. The colony would be made the training school for a variety of outdoor occupations which, after a due probationary period, would come to be a means of livelihood to the individual. At present, in the disposal of patients after treatment, a pressing difficulty occurs because that many of them have no capacity for an outdoor occupation. This would be obviated largely under the scheme which I have proposed.

Length of Residence.

The length of residence must necessarily vary in different cases. It is difficult to lay down a law which is applicable to all. Taking a wide survey of results, however, I incline to fix six months as a desirable minimum. It will be understood, of course, that in some cases this may be curtailed. On the other hand, most of the pronounced disappointments which one has witnessed have been in relation to cases where the hand of the physician has been forced, either through financial or other necessity, or through impatience. To promise or to expect a cure within much shorter limits is apt to effect the discredit of the system.

Digitized by Google

Proportion of Cures.

The question of actual and permanent cure is surrounded with difficulty. In the foreground it is not very easy to define what is meant by cure or recovery. The term is used in different senses by different observers. The term should imply the disappearance of symptoms (including pyrexia), the return to the normal standard of weight (in many cases this is apparently exceeded), the establishment of healthy muscular tone and a stable circulation, the capacity for healthy activity and work of a suitable kind, and the maintenance of these conditions under competent observation for, say, twelve months.

While it is easy enough to obtain this evidence regarding a certain section of patients, the final result in a good many cases eludes observation from one cause and another. This is apt to introduce fallacy into the statement of results unless careful and just deduction be made on this account. I have endeavoured to make a fair computation of the material accessible to me. Without affecting to be absolutely certain, I think that the percentage of satisfactory recoveries may reasonably be expected to range from fifty to sixty if the conditions I have presupposed be fulfilled.

The percentage would be higher were it not for the fact that many cases are sent for treatment at far too late a date, that is to say, at too advanced a stage. This is a deduction which will probably require to be made less in the future, as an intelligent appreciation of the value and limits of the method becomes more general.

The percentage of recoveries will also be increased by the greater effort after early diagnosis, which seems to be already a traceable fruit of the increased attention directed to tuberculosis.

I feel satisfied that were it possible to undertake the treatment of all cases at stages which might fairly be described as incipient, the percentage of recoveries would be remarkably increased. It is difficult, indeed, to see any limit to the proportion of recoveries. In judging of results it is to be kept in view that in addition to the apparently *definite* recoveries, a large percentage of patients show varying degrees of improvement. The percentage of those who go steadily back during the continuance of the treatment is, in my experience, a very small one indeed.

In closing, let me add that it seems to me that we are on the threshold of a still wider development in the application of the open-air method.

The question may well be asked whether we have not, as physicians, been too protective in respect of the treatment of many diseases. With our latter-day conception of the origin of disease, we have gradually relegated the etiological influence of so-called cold to a back seat. In our treatment of disease we want to realise more fully that the action of pure fresh air is directly curative. Within the last few years I have rearranged my conception of practice on these lines, and am fully satisfied with the results obtained.

Digitized by Google

Thus I have treated pleurisy with effusion (I doubt not tuberculous in origin) of such a degree as to require repeated tapping, in the patient's garden, with much comfort throughout to the patient, and with the evident result that the patient manifested far less pyrexic and circulatory disturbance than one usually finds.

In the treatment of the various states which are described vaguely as conditions of delicacy—both in young and grown-up persons—it is remarkable how speedily the indications of ill-health yield to the adoption of fresh air measures. In the adaptation of the system to the treatment of other medical conditions there seems to exist the potential of successes comparable to, if not so striking in character as, those obtained in surgery through the adoption of aseptic measures.

A REPORT ON THE RESULTS OF SANATORIUM TREATMENT IN THE UNITED KINGDOM.

By F. W. BURTON-FANNING, M.D. Cantab., M.R.C.P. Lond.,
Hon. Physician to the Norfolk and Norwich Hospital, England.

The following report deals with 716 patients suffering from pulmonary tuberculosis who have undergone the open-air treatment in certain sanatoria in England, Scotland and Ireland. The medical officers of the following institutions have kindly responded to my request and furnished me with particulars of their patients: Bellevue, Clare, Cotswold, Maitland House, Mendip Hills, Mundesley, Nordrach-on-Mendip, Rossclare, Rostrevor, Rudgwick, Whitmead and Woodburn Sanatoria. Public or charitable institutions for the treatment of tuberculosis have not been included in this inquiry, which has been limited to sanatoria where the inmates defray the cost of their own maintenance and treatment. These payments, however, vary in different establishments from two to six guineas a week, and the records therefore refer to patients drawn from the affluent and from the working classes. There has presumably been a corresponding difference in the style of food, housing, attendance and grounds at the two classes of sanatoria, but I may say at once that the results obtained at them are similar and do not allow of any distinction being drawn between the more expensive and the cheaper sanatoria.

Of the 716 patients, 342 were males and 374 were females. The average age was 28 years, the extremes being 61 and 5 years of age.

Family History.—In 52·2 per cent. of the patients tuberculosis had affected a parent or collateral relative, and in 4 per cent. this tuberculosis was elsewhere than in the lungs.

Contagion.—Proceeding with the inquiry into the etiology of the disease,

Digitized by Google

evidence was next sought in respect of each patient upon the question: Could the occurrence of pulmonary tuberculosis be traced to contagion?

After eliminating all those cases in which heredity might be equally brought forward as the cause, in only 2·6 per cent. of the whole number of patients do the reporters consider that there was satisfactory evidence of the malady having been communicated by another sufferer. The difficulties, however, of arriving at a definite conclusion on this subject of the communicability of the disease are very great, and I am of opinion that the above figures only demonstrate the small number of instances in which exposure to the possibility of contagion is followed more or less directly by the occurrence of the disease. A close inquiry into the past history of these patients convinces me that in a large proportion of them it is impossible to exactly fix the date of the first manifestation of tuberculosis. We learn that prior to the affection for which the patient seeks treatment, he has suffered from a cough which persisted too long to be due to mere catarrh, which was accompanied perhaps by night sweats, by blood-spitting, by unaccountable loss of flesh and strength. These symptoms have, however, eventually passed off, and only in the light of subsequent declared tuberculosis does it become obvious that years ago the patient was already the subject of a tubercular deposit.

Tubercle bacilli emanating from a consumptive patient may gain admission into a healthy person, but years may elapse before, after one or more feints perhaps, the host's resisting powers are broken through and the disease finally established. These intervening years would very much lessen the chance of the malady being ascribed to what was possibly its true source.

Antecedent tuberculosis had been recognised elsewhere than in the lungs in 18·1 per cent. of the patients; 11 per cent. of the whole number had previously suffered from pleurisy; 4·8 per cent. had had strumous glands. In the remaining 2·3 per cent. the bones, joints or peritoneum had been attacked.

In 400 cases especial pains have been taken to ascertain the probable exciting cause of illness, and the following factors receive special prominence:

Influenza was the ascribed cause in	19 per cent.
Overwork was the ascribed cause in	10 „
Insanitary conditions of life was the ascribed cause in	4·2 „
Physical exertion was the ascribed cause in . . .	2 „
Asthma was the ascribed cause in	1·2 „

Passing now to a very brief consideration of a few points in connection with the attack itself:—

Hæmoptysis was present at the onset of the disease in 35 per cent. of these patients and was absent as an early symptom in 65 per cent.

Hæmoptysis occurred during the course of the disease in 37 per cent. and was absent in 63 per cent.

Digitized by Google

In 41·4 per cent. there was no hæmoptysis at any time.

Results of Treatment.—Ninety-two per cent. of all the patients so far responded to treatment as to gain weight. The average gain of weight was 14½lbs.

In no particular do the results of different sanatoria show greater variations than in the matter of the amount of weight added to their patients.

The average gain of weight in one sanatorium was 26lbs., while in more than half the number referred to in this inquiry the average gain was only between 10 and 14lbs.

The number of cases in which the patient's height and previous weight are given are not sufficiently numerous to allow me to deduce any facts which might serve as indications to the amount of weight we should aim at adding to our patients. I do not find any constant relationship existing between the final results as regards recovery or the reverse and the amount of increase in weight.

A fact which is perhaps of equal importance, and which is clearly established by the particulars supplied to me, is that the dietary provided at the sanatoria whose patients show increase of weight above the average does not exceed in fulness the food and milk provided at others. The amount of weight gained clearly does not depend on the mere quantity of food ingested.

Complications.—Concerning the complications met with and their response to the treatment, the following facts are obtained from the information supplied to me.

Laryngitis was stated to be present in 6·2 per cent. of the cases, but I am unable to give the precise nature of the laryngeal affection. In half the cases where the larynx was involved, the pulmonary disease was advanced and the patient did not improve under sanatorium treatment. But of sixteen cases in which laryngitis was associated with slight or moderate lung disease, the aphonia or other manifestation of laryngeal implication was recovered from in no fewer than nine instances.

Tubercular enteritis was noted as existing in 2 per cent. of the cases, and the patients in this group did badly without any exception.

Tubercular peritonitis without obvious disease of the intestinal mucous membrane was observed in two cases, one of whom improved, while the other died.

Tubercular pyelitis occurred in five cases, one of whom improved and maintained his improvement, the others failing to improve.

Fistula in ano complicated the disease while under treatment in five instances; two complete recoveries ensued and two died.

Tubercular disease of the meninges and of the bladder each occurred in a single case.

Pneumothorax supervened in six patients under treatment, two of whom recovered altogether.

There were three patients who developed empyema, and all failed to improve.

Digitized by Google

Asthma was observed in five of the cases, three of whom lost their asthma and also relatively recovered from their tuberculosis. Experience proves that asthma is very markedly benefited by open-air treatment.

Pleurisy was noted as a complication during treatment in twenty-three cases, in three of which it was accompanied by effusion. It did not seem to favour an untoward result, for twelve of the patients made relative recoveries and only two died, the others improving.

Pneumonia, with or without pleurisy, occurred in nineteen patients, three of whom recovered from their tuberculosis, nine improved and seven died.

The average length of stay in the sanatorium has been four months, and the following figures express the final results of treatment:—

Quiescence of the disease or relative recovery was obtained in 37·4 of the patients. *Amelioration* occurred in 40·2 per cent. There was no amelioration in the remaining 22·3 per cent.

The history of these patients only covers a space of about two years or less, and greater value will attach to statistics that may be prepared in the future and relate the after-history of those subjected to sanatorium treatment. At present I can only say that all the patients in whom quiescence of their disease was obtained remain well; of the patients in whom amelioration only occurred, all except 18, or 8·9 per cent., have maintained their improvement.

The opinion has been expressed that women benefit less from sanatorium treatment than men, but this is not borne out by the figures before me.

Of the whole number of women subjected to the treatment, quiescence of the disease or relative recovery occurred in 37 per cent.; in the case of the men the same result was obtained in 39 per cent. Furthermore, an unfavourable issue happened considerably more frequently in the male than in the female inmates, for 26·9 per cent. of the men received no amelioration, against 18·3 of the women.

If we now proceed to classify these cases, certain further information is obtained.

I.—71 per cent. of the patients had fever, an habitual rise of temperature to 99·6° or higher being considered to constitute fever.

II.—8·6 per cent. had rapid cardiac action without fever.

III.—20·4 per cent. had neither fever nor rapid cardiac action.

Class I.—In 50·7 per cent. of the febrile patients, fever disappeared under treatment. I am struck with the fact that in those sanatoria whose records enabled me to work out this point, there is a very close similarity in regard to the proportion of patients whose temperatures responded to treatment. In all the establishments about one-half of the patients admitted with active disease lost their fever. I am not supplied with data enabling me to state the duration of the fever in all these patients, but in my own cases I find that the average time occupied in the reduction of raised temperature

Digitized by Google

to a normal point was 4½ weeks. In a large number of cases with slight and recent fever, a few days sufficed to bring about the fall of temperature, while as much as five months were required in olderstanding cases for the disappearance of fever. From the records before me I find that in no instance did a patient do well who had a sustained temperature exceeding 101·5°, and which could not be reasonably attributed to some non-tubercular or obviously transient occurrence.

Class II.—8·6 per cent. had rapid cardiac action without fever on their admission to the sanatorium, and in addition there were about as many patients whose fever dropped, but whose pulse-frequency did not improve.

Class III.—20·4 per cent. had neither fever nor rapid cardiac action.

Results.

Class I.—Quiescence or relative recovery occurred in 25·1 per cent., and, as we have seen, reduction of fever in 50·7 per cent.

Class II.—Frequency of pulse without fever would appear to be a most unfavourable sign. In only two of the patients did a markedly frequent pulse slow down satisfactorily and allow of the patient's relative recovery.

Class III.—Of those patients without either fever or quickened pulse, 63·6 per cent. secured quiescence of their disease or relative recovery.

I am not enabled from the facts in my possession to classify these patients exactly according to the nature or the extent of their lung disease. I propose merely dividing them into two classes—one class to include all cases where the lesion was limited to one lobe and did not clinically present the signs of cavity, and the other to embrace all cases of more advanced disease.

Class I.—Only 52 patients, or 7·4 per cent. of the whole number, can be described as cases of slight lung mischief. Forty-six, or 88·4 per cent. of those patients with strictly limited disease did well, and attained relative recovery with arrest of their disease. Moreover, so far as a period not exceeding two years in any case allows one to speak, their subsequent progress has been good, for in only two patients has relapse occurred, and one of them again responded well to a second course of treatment.

Concerning these 46 patients, fever was present on admission in 20 of them, 101° being the highest temperature recorded, and, of course, all of them lost their fever. Their stay in the sanatorium extended over four

Digitized by Google

months on the average, and in that time the average increment to the weight was 18lbs.

Tubercle bacilli which had been present vanished from the sputum of 19 of these 46 patients, or in 41·3 per cent. of them. Tubercle bacilli were still discoverable in 16 cases, and in the remainder the point is doubtful, because of the absence of expectoration or other cause.

Some account must, of course, be taken of the cases which, owing either to exceptional virulence of the bacillus or to exceptional weakness of resisting power on the part of the patient, are from the very outset smitten with widespread and serious disease. Early sanatorium treatment may fail to produce any impression on them. But the success attending this treatment in the fortunately more common type of the disease, if only it is commenced soon enough and persevered with long enough, is set forth in the above results, which show recovery and quiescence of the disease in 88·4 per cent. of suitable cases.

I am of opinion that the subsequent records of our sanatoria will contrast favourably with this report. The above account refers to the introduction of this method of treatment into our country. On their first institution the sanatoria had to make up arrears, that is, they admitted confirmed invalids, who would have been placed under this system years previously had the means for its adoption then been brought to their doors, and had it then received the general recognition it now possesses.

The results of treatment must always mainly depend on the selection of cases submitted to it. This point cannot be too often emphasised that the medical man's chief care should be the early recognition of the disease. Let him not wait till his stethoscope discovers the classical signs of excavation, but let him avail himself of all the means for earlier diagnosis of the disease, and let him be urged to treat it sufficiently seriously from the very first.

Though the best results are only to be obtained by early treatment, I should be very unwilling to exclude all advanced cases of pulmonary tuberculosis from sanatorium treatment. In apparently hopeless cases it is an every-day experience to observe an unexpected amount of improvement gradually follow the adoption of the treatment. With the benefit thus gained, and with the lessons in self-management which residence in a sanatorium instils into him, such a consumptive, whose life was fast ebbing away, may be granted a fresh lease for an indefinitely long time, and some powers of usefulness and enjoyment may be gained by his new mode of living.

Digitized by Google

DISCUSSION.

The CHAIRMAN (Sir RICHARD DOUGLAS POWELL): I saw in this room a few moments ago Dr. Dettweiler, who, in association with Brehmer, was amongst the very first to institute the sanatorium treatment of consumption. Unfortunately, Dr. Dettweiler has temporarily lost the use of his voice, so that he is unable to address us this morning, and I think he has been obliged to leave the room, but I am sure we would wish to express our gratitude to him for what he has done. I now call on Professor von Schrötter.

Professor VON SCHRÖTTER (*Vienna*): Ladies and Gentlemen,—I do not think it is necessary to speak now and here upon the usefulness of sanatoria. If there are still some medical men who take an opposite view, and who close their eyes to their advantages, I fear these remarks will not open them. I have brought out the principles in Austria in the sanatorium treatment in a translation of our last report, and I sent a thousand copies over here, but unfortunately they have not arrived, and I cannot give you them; but I hope they will come to-day, and I will try to distribute them as quickly as possible. I say the principles in the treatment of sanatoria are settled, hence there can only be discussed the different peculiarities.

One thing upon which I would lay great weight appears to me—to discern finally, scientifically, whether it is sufficient to keep our patients in our climate here, or if it is necessary to send them still to the south or to the mountains. That question will not be settled until it is observed in a scientific manner, which is not an easy thing to do.

I do not think it is the right way, if any patient comes to you now, to say, "You must go the south, or you must go to the mountains." The conditions are not yet settled sufficiently scientifically.

The plan which we have in Austria, and which we intend to pursue, is as follows: We have our central sanatorium in Alland, near Vienna, at a height of about 400 metres. We have now 130 beds; if we observe some patients for two, three, or four months, and send all of these away in their different peculiarities, I shall send twenty to the south and twelve others up to the mountains, where we shall keep them for a certain time, after which we can say, "These patients have recovered"; and if they show no amelioration I shall have the opportunity of drawing some conclusions.

I should very much wish that we were rich enough to say, "If you would like to send some of your poor patients to such an observation sanatorium as ours, the beds would be at your disposal," but unfortunately we are so poor that we have barely sufficient money for ourselves.

The second thing on which I should lay stress, and what I pronounced already ten years ago, is that in the sanatorium not only must the treatment be climatic, hygienic, dietetic, the free air treatment, but it must be the duty

Digitized by Google

of the sanatorium authorities to study every new treatment and every new remedy which has some founded weight.

At this time certainly one of those remedies is tuberculin. It must not be forgotten that the earth is round. Tuberculin came up, tuberculin went down, and now comes up again. I am sorry to say that I was perhaps one of the first who opposed the tuberculin treatment in tuberculosis, but who said at the same time we must be very thankful to Professor Koch, for his discovery gave us a series of new ideas and points of view. In every respect it is our duty to prove this remedy again, and in a quite independent manner, to become convinced of its real effect. But that appears to be not so easy. We only required a few weeks to know that salicylic acid was a specific against rheumatism, and we only required a very short time to know that diuretin (*theobrominum natr. salyc.*) was an excellent diuretic; but if we require such a long time to know anything with regard to tuberculin the matter must be a more complicated one, and we must try to find the reasons. There is no doubt that Professor Koch wishes to arrive at the truth, just as we do.

I think perhaps the greatest cause of the difficulty is that we have so many different methods in administering this remedy. One person will say that you must give one-twentieth of a milligramme, while others will say you must give twenty milligrammes. One person will say that you can treat every case, and others that you can only take pulmonary diseases, while others say that it is very good for laryngeal, even for abdominal phthisis. What is the truth with regard to the matter? I say we must try to get certain principles and to point out the cases and the manner in which the remedy must be administered. We should be very thankful to Professor Koch if he would kindly help us to state such principles and tell us once more quite accurately how to execute them.

We ought to follow one sure rule, and only afterwards make alterations, in order to obtain certain results with regard to tuberculin. As it appears now, I may say the usefulness of it is no greater than the results obtained by the free-air treatment. If by an average seventy persons in the sanatorium have shown good results from the treatment, the effect of tuberculin appears, according to its promoters, to be equal. But certainly sanatoria are the best places in which to try a new method and get at the truth surrounding it, and only time and experience can bring out the desired results.

Dr. Joh. Dvořák (*K.K. Ober-Sanitätsrath, Spitalsarzt in Opočno-Böhmen*): Hoch ansehnliche Versammlung,—Ich will nur mit ein paar Worten meine Idee, betreffend die Errichtung von eigenen Isolirpavillonen für Tuberkulose in allgemeinen öffentlichen Krankenhäusern zum Ausdruck bringen.

Digitized by Google

ÜBER DIE ERRICHTUNG VON EIGENEN ISOLIRPAVILLONEN FÜR TUBERKULOSE IN ALLG. OFFENTL. KRANKENHÄUSERN.

DR. JOH. DVOŘÁK, *K.K. Ober-Sanitätsrath, Spitalsarzt in Opočno-Böhmen.*

HEUTZUTAGE besteht kein Zweifel darüber, dass die Tuberkulose eine ansteckende und unter Umständen von einem Individuum auf's andere übertragbare Krankheit ist.

Zwischen den einzelnen Fachmännern ist nunmehr noch streitig, in welchem Masse und in welcher Weise die Übertragung und Ansteckung stattfindet (Cornet, Flügge).

Je geringer die Widerstandsfähigkeit des menschlichen Organismus, desto grösser ist die Ansteckungsgefahr durch Tuberkulose. Man kann mit allem Grund sagen, dass die Infectiosität in proportionalem Verhältnisse mit der Widerstandskraft des Organismus steht.

Die Resistenzfähigkeit des menschlichen Organismus nimmt ja durch jede Krankheit mehr-weniger ab, und somit ist die Ansteckungsgefahr bei den in Krankenhäusern behandelten und in einem Saale untereinander untergebrachten Kranken in der Regel am grössten.

Die nächste und natürliche Folge des Nachweises der Infectiosität und der leichten Übertragbarkeit der Tuberkulose, insbesondere auf einen kränklichen und somit weniger widerstandsfähigen Organismus, ist die unbedingte Nothwendigkeit einer strengen Isolirung der mit Tuberkulose behafteten und in den Krankenhäusern behandelten Individuen von den übrigen Kranken.

Bei der bisherigen Einrichtung sind die öffentlichen Krankenhäuser zu einer zweckmässigen Isolirung und zeitgemässen Behandlung von Tuberkulösen nicht geeignet.

Um dem angedeuteten Zwecke zu entsprechen, müssen die Krankenanstalten zum Behufe der Vornahme der Isolirung von Tuberkulösen einer durchgreifenden Reform unterzogen werden.

Zum Behufe einer zweckmässigen Behandlung von abgesonderten Tuberkulösen in eigenen Sonderabtheilungen müssen unbedingt Isolirpavillone, welche den modernen technischen und hygienischen Anforderungen entsprechen, geschaffen werden.

Die Notwendigkeit der Errichtung von Isolirpavillonen für Tuberkulöse in allg. öff. Krankenhäusern erscheint uns durch den nicht unerheblichen Umstand begründet, dass die Ernennung von eigenen Sanatorien nicht nur einen bedeutenden Errichtungs- und Erhaltungsaufwand erfordert, und in denjenigen Staaten, wo die Errichtung und Erhaltung von Lungenheilstätten lediglich auf die Privatwohlthätigkeit angewiesen ist, die Zahl der Sanatorien für die Aufnahme der enormen Summe der minder bemittelten Kranken auf eine unabsehbare Zeit unzureichend sein dürfte.

Digitized by Google

Einen der mächtigsten zeitgemässen Impulse zur Errichtung von Isolirpavillonen für Tuberkulöse in öff. Krankenhäusern dürfte die höchst erfreuliche, durch gediegene Fachmänner und Praktiker begründete Erkenntniss bilden, dass die *Tuberculose auch in der eigenen Heimat*, in einem von atmosphärischen Extremen freien Klima, *mit Erfolg* behandelt werden könne.

Behufs einer erfolgreichen Behandlung der Tuberkulose in dem einheimischen Klima werden neben den Sanatorien die mit den modernen hygienisch-technischen Agentien ausgerüsteten öffentl. Krankenhäuser unschätzbare Dienste leisten. Es hat sich ja in den letzten Jahren die überraschende Erkentniss Bahn gebrochen, dass ein praktischer und anhaltender Erfolg bei der Behandlung der Tuberculose zumeist nur in jenem Klima erreicht werden könne, in welchem der Lungenkranke auch in der Folge sich den Pflichten seines Berufes unterziehen muss. Dies gilt insbesondere von allen Erwerbs- und arbeitenden Klassen. Ausserdem ist der Umstand allein, dass der tuberkulöse Kranke durch die Aufnahme in eine modern eingerichtete Krankenanstalt den schädlichen sanitären Misständen seiner armseligen, und zuweilen inficirten Wohnung entzogen wird, eine nicht gering zu schätzende Errungenschaft für die unteren Bevölkerungsklassen. In der Notwendigkeit einer möglichst wirksamen *Hintanhaltung der Hausinfection* beruht somit auch ein starkes Motiv für die Errichtung von Isolirpavillonen in den öff. Krankenhäusern.

Für die Nothwendigkeit der Errichtung von Isolirpavillonen für Lungenkranke in Krankenhäusern ist auch der nicht minder wichtige Umstand von Belang, dass eine derartige Isolirabtheilung in einer Krankenanstalt zur Aufnahme auch von schweren Fällen der Tuberkulöse geeignet erscheint, welche in den Volksheilstätten nicht aufgenommen werden können. Nach *Schaper* sollten in den Sonderabtheilungen der Krankenhäuser für Lungenkranke schwere Fälle von Tuberkulose für die Aufnahme in die Sanatorien erst vorbereitet werden.

Nach Ansicht einer grossen Anzahl deutscher Fachmänner würden die Sonderabtheilungen für Lungenkranke die Beobachtungs- und erste Behandlungsstationen bilden.

In Deutschland findet trotz der intensiven und hervorragenden Heilstättenfürsorge dennoch die Errichtung von Isolirabtheilungen für Lungenkranke in Krankenhäusern zahlreiche Anhänger und Förderer. Nach *Brehmer* und *Dettweiler* hat sich für die Behandlung der Tuberkulose die Krankenhauspflege als die wirksamste bewährt.

Die Nothwendigkeit derselben wurde von *v. Ziemssen*, *Pettenkofer*, *Schaper*, *Pannwitz*, etc., in Deutschland, und von *Sinclair Coghill* in England anerkannt und nachgewiesen. Sollen unsere öffentlichen Krankenhäuser diesen grossen modernen Anforderungen entsprechen, müssen unbedingt die kompetenten Faktoren allen zeitgemässen Reformen der öff. Krankenhäuser sowol in technischer als auch in hygienischer und therapeutischer Hinsicht Rechnung tragen.

Bei der vorzunehmenden Reconstruction der öffentl. Krankenhäuser müssen insbesondere die wichtigsten Behandlungsfaktoren der Tuberkulöse nach *Brehmer* und *Dettweiler* berücksichtigt werden.

Digitized by Google

Die kardinalsten Forderungen der Isolirpavillone in Krankenhäusern sind somit: freie Lage, Überfluss an gesunder, reiner und freier Luft, leichter Zutritt reichlichen Sonnenscheines, sämtliche Vorkehrungen betreffend die Vorschriften der Antisepsis und Asepsis, und die Durchführung der modernen hygienischdiätetischen Heilmethode. Den modernen hygienischen Anforderungen können lediglich die öffentl. Krankenhäuser auf dem Lande, nicht aber in grossen Städten und in industriellen Centren entsprechen.

Auf Grund meines Antrages und im Sinne meiner Intentionen hat der Landtag des Königreiches Böhmen die Errichtung von besonderen, mit Liegehallen und allen modernen Einrichtungen ausgestalteten Isolirpavillonen für Lungenkranke in allen neu projektirten öffentl. Krankenhäusern in Böhmen zum Beschlusse erhoben.

Nicht genug daran, dass in Böhmen diese Idee durch die genannte gesetzgebende Körperschaft theoretisch verwirklicht, sie fand bereits in vielen öffentl. Krankenanstalten Anklang, und wurde im dem allg. öffentl. Krankenhause in Beneschau im Beginn dieses Jahres mit dem besten Erfolge praktisch durchgeführt.

ON THE ESTABLISHMENT OF SPECIAL ISOLATION BLOCKS FOR TUBERCULOUS CASES IN PUBLIC GENERAL HOSPITALS.

By Dr. JOH. DVORAK,

Ober Sanitätsrath and Hospital Physician, Prague, Bohemia.

There is no longer any doubt nowadays that tuberculosis is an infectious disease which can be communicated from one individual to another. Experts are still divided as to the extent and mode of contagion (Cornet, Flügge). The less the resisting power of the body the greater is the risk of tubercular infection, for the infectiousness of the disease is in proportion to the resistance.

Inasmuch as ill-health lowers the resisting power, the danger of infection must be greatest in the crowded wards of a general hospital. There results from these considerations an absolute necessity for the strict isolation of tubercular cases from others, but under the old conditions most general hospitals have not the arrangements for doing this. Hence the need for the erection of special departments for the isolation of such cases, which departments must correspond to the technical and hygienic requirements of modern science. The necessity for this is all the more pressing, inasmuch as the special sanatoria, both on account of their small number and because of their construction and cost of maintenance, must soon be insufficient for the enormous number of poorer patients. This will be specially the case in those countries such, unfortunately, as Austria, in which the establishment and maintenance of these sanatoria is left to private charity.

One of the most powerful motives for the establishment of isolation

Digitized by Google

pavilions in general hospitals is furnished by the pleasing fact, established both by experts and by practitioners, that tuberculosis can be successfully treated at home, in any temperate climate. In carrying on such cures in the patient's native climate, public hospitals provided with the technical requirements can play a great part. Experience in recent years has shown, in a surprising fashion, that practical and permanent success in the treatment of tuberculosis can for the most part only be attained in that climate in which the patient must afterwards carry on his daily work. This is especially true in the case of the working classes.

Moreover, it is a great gain for the poorer patients to be taken away out of their unhealthy and often pestilential homes into a hygienically constructed institution. The endeavour to combat home-infection is, therefore, another powerful motive for the establishment of such isolation pavilions in general hospitals; the necessity for the latter is further indicated by the fact that they would be able to accommodate those cases of pulmonary tuberculosis which are considered unsuitable for a sanatorium.

Schaper considers that severe cases of phthisis might be prepared in the isolation blocks of chest hospitals for subsequent admission to a sanatorium.

A number of German experts are of opinion that these isolation departments would serve as places for observation and treatment of the disease. In spite of the great vogue of sanatoria in Germany, the movement in favour of isolation departments for phthisis cases is gaining many adherents and advocates.

According to the great experience of Brehmer and Dettweiler, hospital treatment is the most powerful means of rationally combating tuberculosis.

The necessity for isolation pavilions in hospitals has also been recognised by such eminent authorities as v. Ziemssen, Pettenkofer, Schaper, and Pannwitz in Germany, and by Sinclair Coghill in England.

If, however, our public hospitals are to conform to the high claims of to-day, regard must be had, in the most scientific spirit, to all up-to-date reforms, both in construction and in hygienic and therapeutic arrangements. The principles laid down by Brehmer and Dettweiler must be strictly followed. Chief of these are plenty of fresh, pure air, abundant sunshine, adoption of all the rules of modern antisepsis and asepsis, and of the dietetic and hygienic methods of cure. These demands can be met by most public hospitals in the country; not, however, by those in the large towns and industrial centres.

It deserves to be specially mentioned, in conclusion, that the Government of Bohemia has unanimously agreed to establish such isolation pavilions, at all events in all general hospitals which are henceforth to be constructed in Bohemia. These will be provided with "Liegehallen" and all other modern arrangements.

Not only has this idea received the *imprimatur* of the above legislative body, it has already been put to a practical test with the best results in the public General Hospital at Beneschau, and finds a satisfactory echo in many institutions in this country.

Digitized by Google

Dr. JANE H. WALKER (*London*): Sanatorium treatment has deeply interested me for the past nine years. I have treated hospital patients on the open-air method, elsewhere defined by me as a system of elaborated common sense. Good results are apparent and immediate, and successes are very marked at first; later, with more extended experience, difficulties crop up and demand attention. Phthisis is a disease of long and indefinite duration, and it is impossible to tell how long treatment must be carried on. Difficulties arise from the long period of treatment considered from the patients', nurses', and doctors' points of view. Difficulties are greater in paying than in poor sanatoria. In poor sanatoria female patients should do house-work, except cooking and hand-scrubbing; and the men, hedging, ditching, gardening, etc. Better educated patients may act as secretaries, assistant nurses, and do the lighter parts of gardening. The work must be carried out in the absence of strain. Consumptive patients stand strain very badly. They should be watched for a considerable time after leaving sanatoria.

Paying patients are more difficult: they obviously cannot be asked to do house-work, and games of a more or less inane character must be provided, such as croquet, garden golf, bean bags, etc. At the East Anglian Sanatorium we have started sketch and magazine clubs, Greek lessons, painting lessons, courses of reading, etc. Great caution in even pleasurable and amusing excitement is necessary, for it is often fraught with dire results to lung patients.

The nurses' difficulties are from the tediousness of the disease and the unexciting nature of the work. The doctors' difficulties are mainly those of routine.

We are treating patients suffering from one disease, and we must remember we are treating patients as well as the disease from which they are suffering.

Professor E. G. JANEWAY (*New York*): I have only a very few words to say. In the first place, we do not have as yet enough sanatoria, that is, at least, to send all our patients to. What shall we do when these are constructed? I have to meet that problem every day. The patients come to me and ask me, "Doctor, what shall I do?"—whether they are rich, middle class, or poor—and I have to tell them to the best of my ability that they should have fresh air, good food, and be under competent medical attendance wherever they go. And I have seen a good deal of harm result from sending patients into the country, without guarding the people to whom they go against the danger of infection from these patients, and I have thought it would be wise—and I still think it will be wise—on the part of the State of New York to introduce measures which shall take care that the patients that we have to advise to get fresh air will not be sources of infection in country districts where they are not under direct medical supervision. Until we have sanatoria this measure is one of the greatest importance, and I have seen so much harm come both to patients and people to whom they go, from neglect of the precautions given by physicians against the fact that they are liable to be sources of infection, and against the fact that they are liable to infect themselves, that

Digitized by Google

now I make it a rule to refer such patients to someone in the neighbourhood of a sanatorium.

The sanatorium only admits people who cannot afford more than five dollars a week, and charity makes up the rest. Now the rich go there and have cottages, and they are under the same physicians who have charge in the sanatorium, and practically it is an extended sanatorium for the middle class and the richer class, while the poor have the other sanatorium.

The sanatorium was for the wealthier, now they have added a charity addition. There are other sanatoria being constructed, one being at Bedford. The reports of these I will submit; I have not them with me. Now there is another question which has come to my mind as I have listened to this discussion. Where did those nine per cent. of cases of obsolete tubercle get well? Did they get well in sanatoria, or did they get well at home, living the ordinary life? I saw lately, before leaving New York, a gentleman whom I last year told to go to a sanatorium. I told him what measures to adopt. At the end of the year he came back to me to be examined, and practically he was well. He followed the directions I gave him, and he recovered. Ten years ago—I think it was nearer twenty than ten—I made an autopsy of an engineer of a steamship which runs between New York and Cuba. The man had died of yellow fever, and I found in each lung a spot of obsolete tubercle. I asked his family physician, who was there, whether he had had evidence of tuberculosis. He had forgotten. The man's wife said he had had it three years before. Cases do, and can, get well independent of sanatoria. I am an advocate of sanatoria, and I believe in them, but I do not believe until we can get enough sanatoria for the poor we should have sanatoria for the rich. The sanatoria are useful and needful; we want them for the poor who cannot have these things, and for whom I have great sympathy. I am put to my wits' ends to know how to advise. But these facts have come to me. Then there is another which has come to me; it has come to me in my examinations. I have been an examiner for a number of years. The medical practitioner should recognise phthisis in its early stage, and that is one of the greatest defects. The medical profession wants to take more pains to detect early the first signs of the beginning of tuberculosis. More than that, I believe with Professor Brouardel—who yesterday spoke about the method which I advocated a year ago—that we must go out and seek the consumptive in the early stage of his developing the disease, particularly the poor consumptive. We ought to examine patients in all large institutions. That examination should be made by competent people, in order to detect at the earliest possible time the beginning signs of tuberculosis.

Many people come to me with advanced lesions, who have been sick evidently, but do not know what the trouble is. They come—many of them—too late. Then there is one other point: that is, we are not to regard all the lesions of a patient as tubercular or as a mixed infection—that the mixed infection is part of the tubercular process or directly connected with it. I have seen men condemned to death, because in examining the lung

Digitized by Google

the examiner found areas of pneumonia. I had a friend of mine, who had consulted me only a few days before, with tuberculosis, with bacilli in the sputum, and I was told he was about to die with acute tuberculosis. He had fever; he had also areas of consolidation. It was simply influenza with pneumonia. In ten days I told his wife she need not be afraid, he would soon be well. He wanted to go to Colorado. In ten days his fever was gone, his pneumonia was cleared up, and that accounts for so many cases that we meet with, when patients tell us, "Ten or twenty years ago I was condemned to death as a patient with incurable consumption." What has happened is that the patient has been examined by the doctor when he has bronchial pneumonia, which has not been a part of the consumptive process, and from which he recovers, and then he is supposed to have made a miraculous cure. That will explain a great many mistakes we see made, of people who have lost one lung and have recovered, and yet when we make an autopsy both lungs are there.

Sir HERMANN WEBER, M.D. Bonn, F.R.C.P. Lond.: It is with some diffidence that I come before you again. What has been said is so excellent and so exhaustive that it is scarcely possible to add anything. I will confine myself to a very few words.

Dr. Clifford Allbutt asks how long may fever continue.

It is very difficult to say, but I know this, that one case which I have watched very carefully for more than three years, constantly had relapses, and that with rare exceptions his temperature varied between 99°.5 and 103°, and that the patient ultimately recovered completely. Dr. Allbutt mentioned also a subject which is very interesting—*viz.*, that *gout* is a favourable complication. My attention has been attracted by this subject for some years, and I have lately looked up my notes on twenty-five cases which are permanently cured. Amongst these twenty-five cases of complete cure, eighteen have developed very distinct gout, and some of them are now between eighty and eighty-four years of age. Therefore you may say that they have perfectly recovered, although they have still traces of the old disease in their lungs. You ask them, "Do they cough?" They say, "No, only a little phlegm in the morning."

Another remark which has been made by Dr. Fowler is of the greatest importance—*viz.*, about the necessity of "transition sanatoria." When patients leave the ordinary sanatoria, they must have a place where they are for a time under observation and careful management.

As to treatment, you know that the different sanatoria have somewhat different "methods of treatment," but that at most of the sanatoria there prevails a certain "method," which is carried out rather rigidly. As, however, the individual cases differ, they require different treatment, or, at all events, considerable modifications of the usual treatment, and the constitution of the individual in this, as in all other diseases, ought to be taken into first consideration. The "individual" is often forgotten over the "method," with regard to rest, with regard to food, with regard to exercise, and with regard to mental condition.

Digitized by Google

I must now once more crave your pardon. I have been asked to go from this discussion to a little paper of mine which has no intimate relation to our present discussion.

I have called the paper "A Plea for Seaside Sanatoria, especially for the prevention and cure of scrofulous complaints." (See p. 234.)

S. A. KNOPF, M.D. (*New York*): The tuberculosis problem as it stands to-day in the light of the investigations of Koch and his pupils, and in the light of the examples given by Brehmer and by my esteemed and valued friend Professor Dettweiler, it seems to me can be put in a very few words. We cannot solve the tuberculosis problem without a family practitioner. It is to him I look for the solution of the problem first. With regard to the best climate in which to build a sanatorium, opinions still differ. If I was asked what was the best sanatorium for the consumptive labourer, I would say he must be cured in a climate in which he is to live and labour after his restoration to health. If I should be asked what is the best climate for the well-to-do, I would not say Colorado or California, I would not say Nice or Madeira, I would not say Davos, but I would say not too far away from home, so that he will not add to his great misery the other disease so well known as home-sickness. We should seek a climate for the well-to-do, as near as possible, where he can live outdoors the most. That is the whole thing in a nutshell. The more days and nights he is able to enjoy the open air, that is the best climate.

Now to summarise. How should a municipality, how should a Government, proceed in the solution of the tuberculosis problem? And I wish to say here that all which has been said this morning has been said in the interest of the patient. I have not heard one word in the interest of the physician. I am going to say a word in his interest. We have at first to create sanatoria; we all agree in that; not only sanatoria in the mountains, not only sanatoria in the cities, but also, as my esteemed friend Sir Hermann Weber said, we must have seaside sanatoria. We must have sanatoria. We want to cure the mother of tuberculosis as well as the child itself. Although there is an opinion that if a woman afflicted with tuberculosis should become pregnant, she is invariably lost, from that opinion I beg to differ.

At the same time there have been cases on record when a mother taken in time, say six months previous to her confinement, and put in a good sanatorium, with hygienic surroundings, got better, and the baby got well too. So then, in the solution of the problem we must have first, in a large city, for example, a large reception hospital or dispensary.

The far advanced cases should be sent to a special hospital near the city; the better cases should be sent to the mountain sanatoria, if there are any. To do this, to the general hospital should be attached a dispensary to treat patients who go about able to work, but without medical attendance. To these should be added the patients who have been cured, but who attend to avoid a relapse. Now, then, to have a dispensary in a city, to have a city hospital for consumptives, to have sanatoria, to have seaside sanatoria and

Digitized by Google

maternity sanatoria, we must have a large staff of well-trained, well-educated physicians, and these physicians cannot afford to do all this work for nothing. The practitioner who sends away his patient to a sanatorium loses an income, and he has a wife and children to support. I plead that a family practitioner who attends to the dispensary for consumptives should be paid for his services. He cannot afford to go there half a day, and every day, without being paid for doing so. Through the establishment of sanatoria the community will gain. If a patient is treated in time, he will probably get well in six months. In a sanatorium he may cost a dollar a day. If he is not treated, he will become a burden to the community; his family will become a burden to the community, and certainly his loss to the community will be far greater.

Therefore, Mr. Chairman and gentlemen, let us in our eagerness to solve the tuberculosis problem not forget that we must look to the family practitioner in many ways. It is he who sees the consumptive patient first, and it is through his careful examination, through his knowledge of almost every individual in the family, and through his acquaintance with the peculiarities of the individuals, that he will discover an early tuberculosis far earlier than th trained specialist.

I have only one more minute. I will therefore repeat, let us appeal to the State and to municipal governments, let us appeal to our statesmen, let us appeal to our philanthropists, and let us appeal to the general practitioner. Let us have a number of dispensaries in large cities, a number of special hospitals to treat advanced cases. Let us have a Tuberculosis Commission composed of the best elements of the community, laymen and medical men, women and men, to investigate the social conditions of the poor consumptive whom we have taken to the hospital.

We must look out for the poor ones who are left behind, because we have taken the bread-winner to cure. He must have no anxiety that his poor beloved ones are in distress, and only then will we be able to solve the tuberculosis problem.

WILLIAM VICARY SNOW, M.D., F.R.C.P. (*Bournemouth*): Before giving you the results, I will ask you to consider the increased cost to an old institution, the National Sanatorium of Bournemouth, of initiating the open-air treatment of consumption. In addition to structural alterations and the provision of accommodation for an increased staff and the outdoor shelters and accessories, you have at once to double the number of your nurses and employ more servants. No change in the exceptionally liberal diet was required, but each patient costs in food alone *2s. 6d.* per week extra; this is due to the increased power of assimilating food, and especially milk, when spending many hours in the open air.

The statistics I present to you are made by my house surgeons, and give results of treatment from three to six months:—

(1) In the early stages in 63 per cent. we have apparent arrest of the disease, and this percentage would be increased if numbers did not leave at

Digitized by Google

the end of three months to resume their occupations; in 98 per cent. improvement is noted;

(2) In the intermediate stage, 40 per cent.;

(3) In the advanced, 11 per cent.

As far as our inquiries enable us to speak, the improvements established under the new treatment are more permanent. From my own observation, I endorse the statistics of the first stage—the latter, however, appear to me to be rather sanguine.

The following deductions, I hope, will commend themselves to this great meeting:—

(1) That the present hospital accommodation must be rapidly increased;

(2) That consumption should be notified and that every effort should be made by the authorities to induce the patients to apply early for admission.

To those who fear infection, I would say no place is so safe as a well-conducted consumption hospital.

The advantages of sanatoria for paying patients may be briefly summarised:—

(1) The patients learn discipline and how to manage themselves in a more perfect way than they ever would at home;

(2) They can change their residence in accordance with the season of the year; yet in most climates the open-air treatment can be carried out with considerable advantage in residences containing a garden and without the pain of separation from friends.

Ladies and Gentlemen,—From our experience of the rapid diminution of consumption due to improved sanitation, improved wages, and the Factory Acts, I hope I am not too sanguine in predicting that the organised effort now made will be successful, and many of you will live to see the almost entire extinction of a disease which has too often carried off our dearest and best-beloved in the flower of their youth.

Dr. ROSENTHAL (*Copenhagen*): The erection of sanatoria for poor consumptives is very expensive; it will take a long time before sufficient places are supplied for all who need it. Dettweiler estimated nine years ago the number of consumptives in Germany to be 1,200,000. At least it would be required that 400,000 could be treated in sanatoria, which by a treatment of three months would require 100,000 beds. In spite of the energetic efforts in this matter, Germany has up to date only three to four thousand beds for poor consumptives (Helwes). In view of this fact it will be necessary to take care of the consumptives in another manner than by sanatorium treatment, still yet applying the same principles for treatment which are used in sanatoria, with good feeding and fresh air and a suitable hydropathic treatment under the same disciplinary survey as in the closed institutions. As far as I have experienced in the last year, this will be

Digitized by Google

possible in our ordinary hospitals and with very small expenses. Although I estimate the treatment in sanatoria, well situated in a suitable place, to be the best one, I think a good deal of the benefit of the sanatorium treatment can be obtained even in the ordinary hospitals for all those consumptives who cannot be received in sanatoria.

I will shortly refer to you the experiences I have made in the last year in the City Hospital of Copenhagen. I chose a bedroom situated in the great garden of the hospital, as the most free from the dust from the street; the windows were kept open by day and by night. As "Liegehalle," I used a tent in the garden. The patients got six meals a day (maximum expense for nourishment was two shillings a day). Twenty-eight patients were treated—nine in the first, eleven in the second, eight in the third stadium of the sickness (Turban). Most of them (twenty-four) had fever when the treatment began; the average time of treatment was four months. The results were as follows. The fever desisted after shorter or longer time, only two had continued fever during the whole sojourn in the hospital. The average increase of weight for all cases was eighteen pounds in three and a half months (9 kilo.). I will present to you a table of the increase of the weight for each patient. Nine of the treated were dismissed from the hospital without any substantial signs of illness, especially without any cough or expectoration and without râles; only in two of these a few bacilli were found in the early morning by cleansing their throat in this manner; they were all able to do full work. The other nine were dismissed healthy, and also able to do full work; yet there remained with them a little cough and a few râles. For the next six the result was a good one, although not quite satisfactory, inasmuch as they were only partially able to work. The cough and the râles, although greatly diminished, still remained. For the last four the result was as good as nothing.

I need not tell you that the results were the best in the beginning cases, with only small affections of the lungs, and that I have avoided undertaking to cure hopeless cases. You will see from the table presented that all the treated increased in weight, even if the final result was little or none. Therefore I have not estimated the benefit of the treatment only, or even for the most part, from the increasing of the weight.

1ST STADIUM.

1	...	...	4,800	grammes	...	...	1½ months
2	...	...	11,250	,,	...	...	2½ ,,
3	...	...	5,000	,,	...	...	1 ,,
4	...	...	4,200	,,	...	...	4 ,,
5	...	...	11,950	,,	...	...	4¾ ,,
6	...	...	8,500	,,	...	...	3 ,,
7	...	...	9,900	,,	...	...	2 ,,
8	...	...	19,650	,,	...	..	4¼ ,,
9	...	...	3,450	,,	...	...	1½ ,,

Average of increase ... ... 8·75 kilo. in 2·7 months.

Digitized by Google

2ND STADIUM.

1	... ...	1,800 grammes	...	...	½ months
2	... ...	3,400 ,,	...	...	2½ ,,
3	... ...	7,250 ,,	...	...	2 ,,
4	... ...	11,700 ,,	...	...	5 ,,
5	... ...	14,500 ,,	...	...	4½ ,,
6	... ...	17,700 ,,	...	...	4 ,,
7	... ...	9,100 ,,	...	...	4 ,,
8	... ...	16,250 ,,	...	...	5 ,,
9	... ...	7,500 ,,	...	...	5½ ,,
10	... ...	13,300 ,,	...	...	3½ ,,
11	... ...	8,500 ,,	...	...	2 ,,

Average of increase ... ... 10·1 kilo. in 3·5 months.

3RD STADIUM.

1	... ...	4·500 kilo.	...	...	3½ months
2	... ...	11·300 ,,	...	...	6 ,,
3	... ...	20·700 ,,	...	...	5 ,,
4	... ...	7·800 ,,	...	...	4½ ,,
5	... ...	5·100 ,,	...	...	2½ ,,
6	... ...	4·900 ,,	...	...	3½ ,,
7	... ...	8·400 ,,	...	...	4½ ,,
8	... ...	8·50 ,,	...	...	3 ,,

Average of increase ... ... 7·94 kilo. in 4 months.

Average of increase of all (28) treated cases ... 9·0 ,, ,, 3·4 ,,

AVERAGE OF WEIGHT-INCREASE AFTER THE RESULT.

For 9 "relatively healed" cases	...	...	9·94 kilo.
,, 9 "much improved" cases	...	...	11·13 ,,
,, 6 "improved" cases	...	...	7·5 ,,
,, 4 "not improved" cases	...	...	3·7 ,,

What I intend by this short communication is only to ask my colleagues to proceed in the same manner and try to effect sanatorium principles in ordinary hospitals, especially those which are situated in an open country, with plenty of fresh air and free from all the dust of the town. I am sure you will do the best possible in awaiting the erection of sanatoria, and you may be able to do it with only small expenses.

Dr. BURTON-FANNING said he had already had the privilege of addressing the members of the Section in the report on "Results of Sanatorium Treatment in England" placed in their hands; he would therefore limit his remarks to an expression of his appreciation, from the point of view of a sanatorium worker, of the remarks made by Professor Allbutt and others. He was glad that the opener did not attach much importance to conditions of weather. He said equally good, if not better, results were obtained in the winter as in the summer, that the chief difference lay in the comfort with which the treatment could be carried out by the patients. It was somewhat surprising to find with what success the system could be followed, even in a town hospital. He agreed with Dr. Philip that no limitation need be placed on the exposure

Digitized by Google

of the patient to open air, provided only that he was protected from wind, rain, and sun.

Rest, he thought, as far as one constituent could be placed above others, was of greatest importance. His practice was to begin the course of treatment of most patients with absolute confinement to bed, and the first few weeks of complete repose, together with the fresh air and feeding, usually decided the amenability of the case to treatment. The necessity of rest also is the most essential thing to impress on the patient when he leaves the sanatorium. Over-fatigue is the cause of relapse more commonly than any other one factor.

As regards the persistence of fever in cases that eventually do well, I would say that if encouraged by a progressive, though slight, lowering of fever week by week, we should persevere with treatment for many months. Cases that show no lowering after one month will probably not benefit materially.

As regards diet my practice is to decide what weight should our patient be brought up to—taking into consideration the patient's previous maximum weight and his height—and then, by watching the weekly records, to vary the diet both in quantity and quality according to requirements. So necessary is it to always study the digestion that one often gets greater gain of weight with reduced diet than with increased diet.

Hydrotherapy and massage are apparently not much used here. Personally I have tried the effect of massage and resisted movements in those unfavourable quick pulse cases, and have been disappointed at the results.

Dr. WOLF BECHER (*Berlin*) and Dr. RUDOLF LENNHOFF (*Berlin*) made a joint communication on "Erholungsstätten" (abstract):—

Only a small number of consumptive patients can be admitted into sanatoria. Many, who are to be admitted, have to wait so long, that in the meantime the disease becomes serious. Scarcely anything is done for such patients, who cannot be admitted into sanatoria. Those discharged from sanatoria in many cases recommence work too quickly, instead of passing through an intermediate stage. The "Erholungsstätten" serve a useful purpose in all these cases.

The State, municipalities, or private owners place a piece of forest land near large centres of population at the disposal of the institution; this land is wired in. *Arrangement:* Döcker's barrack with kitchen, office, room with one to two beds for urgent cases until removal into a hospital. A hall—quite open on one side—for bad weather. Folding chairs are provided and games are encouraged.

The patients travel there in the morning and return to their own home at night. They receive milk, dinner, and other food.

In Germany most of the people in question are insured against sickness (by Krankenkassen). These Krankenkassen provide support towards maintenance of families, payment of railway fares, and cost of milk and dinner (in Berlin 9–10 pence per day).

Digitized by Google

A medical man superintends the institution, but every patient remains under the treatment of his own doctor. The institution is under the management of a nurse. There are also a cook, a scullery-maid, and a night watchman. The institution makes a profit upon the sale of the milk, which suffices to pay the nurse and the servants.

The prime cost of the whole institution is only about 6000 to 7000 marks (but if, as in Berlin, the "Red Cross" lends the Döcker's barrack, the cost only amounts to 3000 to 4000 marks).

In order to test the practicability of our scheme, the German "Red Cross," in the summer of 1900, established the first Erholungstätte (for men only) in the Jungfernhaide, a forest near Berlin. In consequence of the good results of the first year's working, this summer the "Red Cross" established four Erholungsstätten, two for men, and two for women, each for 150 patients. Large manufactories (for their own workmen only) and other towns have also followed suit.

ARZT WOLF BECHER (*Berlin*): Meine Damen und Herren,—Ich möchte mir erlauben, Sie ganz kurz auf eine neue Art von Sanatorium aufmerksam zu machen. Es handelt sich um Sanatorien für Lungenkranke, die die Eigenheit haben, dass sie nur über Tag den Kranken zugänglich sind; über Nacht halten sich die Kranken in ihrer Behausung auf.

Ueber den Weg, auf welchem ich zu diesem Sanatorien gekommen bin, möchte ich mir erlauben, Ihnen ganz kurz zu berichten. Mein Freund, Dr. Lennhof, und ich haben i. J. 1899, in der Zeit als der Tuberkulose-Congress in Berlin in Vorbereitung war, eine Reihe von Untersuchungen angestellt darüber, wie die Lungenkranken wohnen, wie sie sich in ihrer Wohnung verhalten, wie sie den Tag zubringen. Dabei waren wir in der Lage festzustellen, dass es mit der Wohnung der Kranken überaus schlecht bestellt ist. Wir fanden die Lungenkranken, wie das in allen grossen Städten der Fall ist, z. T. in Hintergebäuden, z. T. in hoch gelegenen Geschossen, wo sie durchweg in Verhältnissen leben, die durchaus dem widersprechen, was den Lungenkranken zuträglich ist. Weiterhin haben wir untersucht, wie die Lungenkranken den Tag verbringen. Ueberall wird dem Lungenkranken der Ratschlag gegeben, er soll möglichst viel im Freien sich aufhalten. Ich will gleich hinzufügen, dass auch bei uns, trotz der grössten Bemühungen um die Errichtung von Sanatorien, der allergrösste Teil der Lungenkranken nicht in die Sanatorien aufgenommen werden kann. Wenn wir auch Tausende von Betten jetzt bereit haben, so bleibt doch noch ein grosser Teil von Lungenkranken der nicht aus seiner Behausung heraus zu schaffen ist, der gezwungen ist, in seiner Behausung zu bleiben, und wie jeder andere Patient im Hause behandelt wird. Als wir untersuchten, wie diese Kranken die Weisung des Arztes befolgen, möglichst viel in's Freie zu gehen, fanden wir, dass Alles, was sie thun konnten, darin bestand, dass sie entweder auf der Strasse spazieren gingen oder die nächsten Plätze aufsuchten, insgesamt aber lebten sie stets in der Stadtluft.

Nachdem wir dies festgestellt hatten, erwuchs mir persönlich das Problem:

Digitized by Google

Was kann geschehen, um grössere Mengen von Tuberkulösen ohne viele Ausgaben, ohne viel Geldaufwendungen, über Tage aus der Stadt heraus in's Freie zu bringen? Dieses Problem zu lösen, ist mir gelungen, u. z. unter Zuhilfenahme von verschiedenen Einrichtungen, die spezifisch deutsch sind. Ich muss noch hinweisen auf unsere soziale Gesetzgebung, unsere Arbeitergesetzgebung. Es wird einem grösseren Teil von Ihnen bekannt sein, dass wir in Deutschland ein Krankenkassengesetz haben, dessen Wirkung darin besteht, dass für einen jeden Kranken Arznei und ein gewisses Krankengeld stets zur Hand ist. Diese Kassen beruhen auf Gegenseitigkeit, sind aber gesetzlich eingeführt, sodass ein jeder Arbeiter, der über ein gewisses Minimum von Einnahmen verfügt—bis zu 1500 Mark—der gesetzlichen Versicherungspflicht unterliegt. Wir nehmen zunächst diese Krankenkassen zu Hilfe, indem wir sagen, die Krankenkassen haben die Verpflichtung, für die Kranken zu sorgen. Wir können also das Krankengeld, das die Kranken bekommen, auch für unsere Idee verwerten, für die Ernährung der Kranken, wenn sie in Heilanstalten untergebracht sind.

Die Idee, die ich hatte, war folgende: Man errichtet im Freien, im Walde, Baracken, wie wir sie vom "Roten Kreuz" haben, und schafft die Kranken dorthin hinaus. Sie bleiben dort über Tage, den ganzen Tag hindurch, werden dort beköstigt und gehen am Abend in ihre Wohnungen zurück, am nächsten Morgen wieder hinaus und so fort, solange es ihnen beliebt und es nötig erscheint. Ich möchte darauf hinweisen, dass der springende Punkt in der ganzen Sache die ungemeine Billigkeit der Einrichtungen, sie heissen *Erholungsstätten*, ist. Wir haben mit einer Erholungsstätte begonnen. Hier muss ich mit grossem Dank der Mitarbeit, ja führenden Arbeit, des Herrn Dr. *Pannwitz* gedenken, der Ihnen Allen als Tuberkuloseforscher bekannt ist, gedenken. (Beifall.) Es war Herr Dr. Pannwitz, der sich der Verwirklichung der Erholungsstättenidee angelegen sein liess, und mit allen Mitteln darauf drang, dass wir bereits in 1900 den ersten Versuch machen konnten. Darüber ist nicht viel bekannt geworden. Ich erlaube mir, Bilder herumzugeben, die Ihnen unsere Einrichtung zeigen. Die Kranken gehen des Morgens hinaus und halten sich im Walde auf. Sie sehen auf dem ersten Bilde die Kranken, wie sie sich wie in allen anderen Sanatorien im Walde aufhalten. Ich muss hinzufügen, dass das Gelände uns nichts kostet; wir bekommen es umsonst. Die königlich preussische Forstverwaltung hat sich von vornherein bereit erklärt, unser Unternehmen zu unterstützen, und giebt uns den Grund und Boden umsonst her. Weitere Unterstützungen haben wir von anderen preussischen Behörden. Es handelt sich darum, die Kranken für billiges Geld weit hinauszuschaffen in's Freie. Unsere Erholungsstätten liegen vielleicht $1\frac{1}{2}$ bis 2 Meilen von der Stadt entfernt in der besten Luft. Die Königliche Eisenbahn hat uns die allergünstigten Bedingungen für unsere Kranken gewährt; für einen Spottpreis, für ein paar Pfennige kommen sie Tag für Tag hinaus.

Nachdem die Kranken dort hinausgekommen sind, werden sie verpflegt, sie erhalten Milch,—Sie sehen auf diesem Bilde die Vertheilung der Milch; auf einem weiteren Bilde sehen Sie die Kranken bei ihrer Unterhaltung.

Digitized by Google

Wir haben hier zwei Baracken errichtet, die wir vom "Roten Kreuz" geliehen bekommen. Die eine dieser Baracken wird als Wirtschaftsraum und Bureauraum verwendet, die andere als Sommeraufenthalt benutzt. Auf einem weiteren Bilde haben Sie Gelegenheit, die Vertheilung des Mittagessens zu sehen. Ich betonte schon vorher, dass das Wesentlichste die Billigkeit der Einrichtung ist. Eine ganze Erholungsstätte kostet nach deutschem Gelde etwa 3–4000 Mark, und in dieser einen einzigen Erholungsstätte sind wir in der Lage, 150 Kranke täglich zu beherbergen. Diese ganze Einrichtung war nur deswegen möglich, weil wir von vornherein darauf verzichteten, die Kranken über Nacht dort zu behalten. Wir haben einen Arzt, eine Schwester vom "Roten Kreuz" oder eine Johanniter-Schwester und zum Wirthschafsbetrieb mehrere Wirthschafterinnen. Wir haben die Sache derart hergestellt, dass der Betrieb der ganzen Erholungsstätte sich selbst deckt. Das kommt so zu Stande, dass wir die Milch im Grossen ankaufen und zu dem in Berlin üblichen Einzelpreis verkaufen. Dabei entsteht ein Nutten, aus dem bezahlt werden können die Schwester, die Wärterin u. s. w. Nur für den Notfall haben wir für plötzlich Erkrankte zwei Betten aufgestellt, die nur überaus selten in Gebrauch genommen werden.

Im Jahre 1900 machten wir den ersten Versuch mit der Errichtung einer Erholungsstätte in der Jungfernhaide. Es sollte lediglich erprobt werden, wie die Dinge in der Praxis sich gestalten würden. Dieser Versuch ist gelungen, und jetzt sind wir über das Versuchsstadium weit hinaus. Wir haben jetzt ausser in der Jungfernhaide eine zweite Erholungsstätte in Pankow errichtet für Frauen und Mädchen. Eine dritte ist von der Allgemeinen Elektrizitätsgesellschaft errichtet worden. Zwei weitere sind in Bau. Wir haben also in Kürze in Berlin fünf Erholungsstätten im nächsten Umkreise, und wir werden im Stande sein, 600–700 Kranke zu gleicher Zeit zu verpflegen.

Ich habe noch einige Worte hinzuzufügen über die Art der Kranken. Unsere Erholungsstätten unterscheiden sich von allen anderen dadurch, dass wir keinerlei Bedingungen an die Aufnahme knüpfen. Wir nehmen einen jeden Kranken auf, der uns von seinem Arzt zugeschickt wird. Die Kranken bleiben auch während des Aufenthalts in unserer Erholungsstätte in der Behandlung ihres Arztes; nur Noth-Hilfe wird geleistet. Die Kontinuität von Arzt und Kranken wird durchaus nicht unterbrochen. Der Kranke hat Gelegenheit, so oft es ihm beliebt, zum Arzt zu gehen. Dann kommt er eben eine Stunde später in unsere Erholungsstätte. Auch der Arzt hat Gelegenheit, seine Kranken jederzeit aufzusuchen; er hat nur einen Weg von einer Stunde in unsere Erholungsstätte zu machen. Da wir keine Beschränkung in der Aufnahme der Kranken haben, so haben wir einmal frühe Fälle der Tuberkulöse; wir bekommen Patienten, bei denen z. T. noch die Tuberkulose latent ist. Anderseits haben wir die spätesten Fälle von Tuberkulose mit schwerer Larynxpthise—solche Kranke, die schon eine längere Kur durchgemacht haben, aber zu den Unheilbaren gehören. Wir haben in Deutschland die grösste Not, für derlei Kranke Rat zu schaffen. Für die Sommermonate leisten zweifellos die Erholungsstätte auf diesem

Digitized by Google

Gebiete etwas ganz Besonderes. Zweitens macht sich die Erholungsstätte sehr nutzbar für solche Kranke, die für feste Lungenheilstätten vorgemerkt sind. Es ist Ihnen bekannt, dass trotz der Menge der Volksheilstätten, die wir haben, der Andrang ungemein gross ist. Es ist nicht zu vermeiden, dass Kranke oft monatelang warten müssen, bis sie aufgenommen werden können. Das hat seinen Grund darin, dass in Deutschland auch im Volke die Meinung sich schon festgesetzt hat, dass die Lungentuberkulose zu den heilbaren Krankheiten gehört und dass auch die ärmsten Kranken sich bemühen, mit Hilfe der Versicherungsanstalten und der sozialen Gesetzgebung Aufnahme in Heilstätten zu erhalten. Wir behalten die Kranken, die in die festen Lungenheilstätten nicht aufgenommen werden können, so lange, bis sich ihnen die Pforten der Lungenheilstätten öffnen. Das gestaltet sich so, dass die Kranken z. B. am Mittag oder am Abend unsere Anstalt verlassen und dann zur Bahn gehen, um sofort in feste Lungenheilstätten aufgenommen zu werden.

Anderseits haben wir auch schwer Tuberkulöse. Wir haben unter unseren Kranken grosse Gruppen von solchen und müssen nach unserer Erfahrung darauf dingen, dass überall, wo sich Siechenhäuser für Tuberkulöse noch nicht befinden, durch Erholungsstätten ungemein viel gethan werden kann. Man muss sich wundern, mit welchem Triebe von Selbsterhaltung die schwer Tuberkulösen sich noch dann Erholungsstätten zuwenden; sie scheuen den Weg nicht, sie scheuen die Mühe nicht, die ihnen daraus erwächst, dass sie einen Weg von ½ bis 1 Stunde zu machen haben.

Sie werden einsehen, dass die Erholungsstätten bei dem losen Verhältnis zu den Kranken nicht zu vergleichen ist mit festen Sanatorien mit Bezug auf die Resultate. Wir verzichten darauf, jegliche Untersuchungen an den Kranken anzustellen; wir beschränken uns auf die Leitung und Überwachung. Wir nehmen nur an den Kranken Wägungen vor und lassen uns von dem behandelnden Arzt Bericht erstatten. So findet ein gemeinsames Arbeiten zwischen dem Arzt der Erholungstätte und dem Arzt der Patienten statt. Trotz aller erschwerenden Bedingungen lässt sich aber doch erkennen, dass die Erholungstätten überaus wirksam sind. Wir haben feststellen können, dass nicht unbeträchtliche Zunahmen an Gewicht zu verzeichnen waren. Freilich aber haben wir auch Kranke genug, die immer weiter ihrem gewissen Verhängnis zugehen; insgesamt aber müssen wir sagen, dass mit den Erholungsstätten ein wesentlicher Fortschritt in Bezug auf die Tuberkulose-Behandlung bei uns erreicht worden ist. Wir sind der Ueberzeugung, dass diese neue Einrichtung aus dem Versuchsstadium heraus ist, und hegen den dringenden Wunsch, dass auch in anderen Staaten mindestens Versuche gemacht werden in derselben Weise, wie wir es in Berlin gemacht haben. Vor allem ist die Errichtung von Erholungsstätten notwendig für Fabrikstädte und Grossstädte, wo es unmöglich ist bei den jetzigen Verhältnissen, irgendwie Bedingungen zu schaffen, unter denen ein Lungenkranker, den wir nicht im Sanatorium behandeln können, in der Besserung fortschreiten kann.

Ich möchte schliessen, in dem ich dem Wunsche Ausdruck gebe, dass das Ausland unsere neue Einrichtung prüfen möge, und in dem ich noch die

Digitized by Google

Hoffnung ausspreche, dass Sie auch wie wir einen Erfolg und eine Freude daran haben werden. (Lebhafter Beifall.)

W. R. HUGGARD, M.A., M.D. (*Davos Platz*): There are some points in the question before us on which we are probably all agreed. There is probably no difference of opinion amongst us as to the general principles. We all think that good food and enough of it, fresh air with rest and exercise according to the patient's condition, are absolutely necessary in the treatment of pulmonary tuberculosis. We are further agreed that care is necessary to prevent the patient from becoming a source of infection to others.

Now can these objects be attained only, or best attained, in a sanatorium? Is a sanatorium the best place for every case? For some cases, yes; but certainly not for all. For all those people who are unable otherwise to secure suitable attention, hygienic arrangements and medical supervision, a sanatorium is obviously the best place. It is also the best place for the wilful, the thoughtless, and the impulsive. The great advantage of sanatorium life is that the patient is drilled into the practice of hygiene, and his task is made easier by having fellow-learners in the same discipline. He is made to take sufficient food, to rest, and to exercise according to his condition; and he is more likely to have answered his daily prayer not to be led into temptation. In a sanatorium too the patient is under the immediate eye of his doctor; and the beginnings of new ailments are seen to at once. Patients suffering from laryngeal tuberculosis are also, as a rule, best treated in a sanatorium, where their tendency to talk too much can be kept under control.

In many cases, however, the cast-iron discipline of a sanatorium is unnecessary; in many others unavailing. Where the needful hygienic and general conditions can be secured with the intelligent co-operation of the patient, all the requirements of treatment can be carried out without recourse to the rigid system of a school. In some cases also it must be recognised that one ending only to the disease is inevitable. When this is the case little is to be gained by drilling the patient. Discipline is out of place, and, except occasionally, the thermometer does not then afford information to compensate for the anxiety or the inconvenience occasioned by its use.

Another consideration should weigh with us in determining whether a case would be better in a sanatorium or outside. The psychical element is important in the treatment of tuberculosis. Moping, worry, or depressing mental states of any kind have a prejudicial influence on the course of the disease. One great object of treatment then, in most cases, will be to place the patient where there will be greatest freedom from worry, fretting, irritation, depression or excitement. This object may or may not be best secured in a sanatorium. No general rule can be laid down; but the point should receive full attention before we decide what is best for the individual patient. A patient will commonly make better progress in hygienic conditions short of the best than in ideal surroundings with worry or other depressing mental influence.

Digitized by Google

Just a word in regard to one or two points on which great stress is laid in some sanatoria: increase in weight and over-feeding. Increase in weight, though a good sign when joined with improvement in other respects, is not by itself necessarily an index of improvement, much less of recovery. In many a case the disease steadily advances when at the same time the patient is putting on weight, while cases are not infrequent in which recovery takes place though the patient remains thin.

Over-feeding, useful as it may be in some instances, certainly does harm, like over-doing anything else, when injudiciously enforced. The aim of treatment should surely be to attain the highest level of nutrition; but over-feeding by no means always secures this result.

In medicine, as in other affairs, a hobby or a system may be ridden to death; and I have not been able to assure myself that sanatoria generally are free from this tendency. A cynic once remarked that while there was a great deal of difference between a good doctor and a bad doctor, there was very little difference between a good doctor and no doctor at all. Does the same difference exist between different sanatoria?

There is a tendency on the part of some to exalt the sanatorium system, to make it appear that climate and other considerations are of little importance, that any person of ordinary intelligence can carry out the system satisfactorily. I am inclined to think, on the contrary, that the man is more important than the system. A good man will always get good results; while the best system in the world applied zealously but without discretion will fail of its purpose. No system has yet been devised that will replace common sense. Rules are a poor substitute for principles. The conscientious and rigorous employment of any system will not atone for lack of clinical insight and for want of judgment. Is it not possible that the head of a sanatorium may lack firmness or may lack discretion, that he may not himself be gifted with a well-balanced and judicious mind? Sometimes a sensible patient might be less likely to do foolish things if left to his own judgment than if directed by a well-meaning but impulsive or injudicious autocrat.

I do not therefore think, as is now to some extent the fashion, that the whole duty of man is to send every case of phthisis to a sanatorium.

Nearly every patient would indeed be better for residence in a good sanatorium, for some time at least. A course of sanatorium treatment has much the same educational value on the grown patient as the kicking and the cudgelling a boy goes through at school, and I must confess that no patients are so easy and pleasant to deal with as those who have gone through this preliminary discipline in a sanatorium.

There are one or two points on which I think misleading ideas have gained currency. I refer to sanatorium "cures" and the comparative results of sanatorium and of outside treatment.

The term "cured" is sometimes used in rather a loose fashion. I have known the expression applied to a patient whose expectoration still contained tubercle bacilli, and had not diminished in amount. The patient had indeed gained in weight, in strength, and in general health, and was able to return

Digitized by Google

to business. The term was used in this way not by the patient, but to me personally by the medical man himself. When gently bantered on his use of the term he withdrew it, but evidently he sometimes used it in rather a misleading sense.

There is another source of error in the interpretation of sanatorium statistics. Some sanatoria will not accept severe cases. They take, in fact, only promising cases. A large percentage of recoveries obviously has not the same meaning in selected as in unselected cases. If a sanatorium has more applicants for admission than it can receive, I see no reason why it should not give the preference to those whom it thinks it can help most. The practice is perfectly legitimate, but where followed—for it is not, I believe, adopted by all sanatoria—it would modify, to some extent, our interpretation of the statistics.

In conclusion, may I quote the best statistics as to recovery in pulmonary tuberculosis with which I am acquainted? They are free from the suspicion that their author was pleading for his own house. They come from the Morgue in Paris. Vibert, in looking over the register of the necropsies made at the Paris Morgue "was struck by the fact that in one hundred and thirty-one individuals of from twenty-five to fifty-five years of age, having all succumbed to violent or sudden deaths, it was noted that the existence of pulmonary tuberculosis was recognised in twenty-five, in seventeen of whom the malady was in a cretaceous or fibroid state—that is to say, of tubercles cured." In other words, the disease was present in 19·08 per cent. of all cases, and in 68 per cent. of those in whom it was present it was cured.

Any sanatorium might be proud of such results, but the figures may afford hope and consolation to a good many of us. A considerable proportion of cases will get well under the worst system and the most incompetent advice.

C. BRAINE-HARTNELL, M.R.C.S. Eng. (*Cotswold Sanatorium*): There is a difference of opinion as to where temperatures should be taken, the mouth, axilla, or rectum. The majority of men are opposed to rectal temperature, but it is absolutely necessary in patients living in the open air. The æsthetic reason for not taking a rectal temperature should not weigh with us too much if we are to do our patients justice. If we are to regulate a patient's actions for the whole day by the temperature chart, this chart must be an accurate one.

The oral records are unreliable. Three charts are shown illustrating this point, in all of which the temperature never rose above normal, though patients had active disease.

With regard to the limits allowed in rectal temperatures, the temperature should be normal morning and evening, and if taken directly after exercise, should generally be about 100°, though this is a matter of idiosyncrasy. If temperature be taken one hour after exercise, it will usually be under 99°.

Many charts are shown illustrating the difference between oral and axillary records.

It is in the lower ranges of temperature that the difference is most marked,

Digitized by Google

ALFRED R. PARSONS, M.D. (*Dublin*), referred to the prevalence of tuberculosis in Ireland, and mentioned that it causes an annual mortality of over 12,000, which is practically one-seventh of the total mortality. The deaths from tuberculosis surpass by one third the deaths caused by all the zymotic diseases. Notwithstanding this appalling death-rate, no special effort was made in Ireland to cope with it till within the last few years, when the Forster Green Hospital was opened, near Belfast, and the National Hospital for Consumption at Newcastle, co. Wicklow, some twenty-five miles from Dublin. From the opening of the latter hospital on the 19th March, 1896, to the end of last December, 423 patients were treated and discharged. Of these 80 were very much improved, and many afford a strong probability of complete arrest; 129 were much improved, and 136 were improved.

None of the cases are returned as "cured." As to the mind of the speaker, only those cases which stand the test of time should be so classified, and he did not feel that sufficient time had yet elapsed to apply that test, but he could say that cases after their discharge from Newcastle had passed into the Army, Post Office, etc. He drew attention to a chart prepared by Dr. Steede, which showed the average number of ounces gained by each patient per week for each month of the four years since the hospital was opened. It could be seen that the least gain in weight took place during March and June, and the maximum gain in September, October, November, and December.

In his opinion sanatorial treatment was necessary for rich and poor. Theoretically, the medical profession might believe in the fresh-air treatment, practically they lived as if they did not believe in it, and consequently it would not be probable that patients would carry out the open-air treatment at their homes. Sanatoria were still more necessary for the poor, but they must not be allowed to return to their wretched hovels, but sent to a "Colonie" in the country. At the very foundation of the prophylaxis and cure of consumption is the problem of the better housing of the poor.

HOWARD SINCLAIR, M.D. (*Rostrevor*): Having for the past thirteen years been connected with the Hospital for Consumption of Belfast, a city having the unenviable reputation of a death-rate of 29 per 10,000 from pulmonary tuberculosis, I was able to form an estimate of the advantages of the change from "hospital" to "sanatorium" treatment. I had intended to give some brief account of my experience in connection with the Forster Green Hospital, but as so much of your time has already been occupied, I shall content myself by simply referring to a few points, some of which have been mentioned by previous speakers. That climate is of comparatively small importance *per se* in the absence of care, four cases during the past twelve months in my own sanatorium at Rostrevor are confirmatory evidence. One, a lady who two years ago went out perfectly healthy to Egypt, after living in the desert one year, came home three weeks ago with well-marked pulmonary tuberculosis; two others developed pulmonary tuberculosis in South Africa; and a fourth, at sixty years of age, developed the same disease in West Australia.

Digitized by Google

Dr. Dettweiler many years ago impressed me by pointing out what he considered the two most important factors in successful treatment of phthisis: first, getting the patient early; second, treating him in his own climate.

Dr. Clifford Allbutt very properly referred to the unreasoning system of indiscriminate stuffing, which seems to have been adopted in so many British sanatoria, with the unfortunate danger of bringing sanatorium treatment into disrepute. I have one case who is recovering from three months' treatment of this sort, which had produced dilatation of the stomach and a complete distaste for food.

I have for a long time allotted gardens to those who fancied it, but I find photography is really the amusement which is most popular with patients.

The treatment of the individual is really what we must aim at. It is the soil on which the bacillus grows rather than the germ itself which we must combat. On that account the mental state of the individual must be taken into account. A patient inclined to be despondent must not be too much isolated, and the easily excited one should not be placed in the neighbourhood of a talker.

A point in connection with the site of a sanatorium, too often overlooked, is local shelter. An excellent view will not compensate for a closed window in case of certain winds, and if the local situation allows of glass being practically abandoned altogether, as I have fortunately been able to do, even in winter gales, so much more fresh air is gained for one's patients.

In treating patients of the working classes in numbers it is, I believe, necessary, in order to have proper supervision of rest and exercise, to do so by means of verandahs; and in a climate such as ours one verandah is insufficient unless the site of the sanatorium is so perfect that it is protected from all strong winds. The aphorism that strong winds are poison to the consumptive cannot be too strongly insisted upon. Therefore if at least one auxiliary verandah with a different aspect can be provided, it prevents the necessity for drawing curtains across its front, a practice which has a most depressing, chilling effect on those who are unfortunate enough to be placed behind it.

Local situation is also one feature that appears to me very often neglected in the choice of a site. For instance, in one sanatorium, perfect in many respects, I found nearly all the windows closed because wind and rain were driving in. This is bound to be the case in sites selected with the idea of having extensive views from the bedroom windows. By all means let us have the outlook bright and cheerful, but don't adopt it at the expense of closed windows.

Early in 1899 I succeeded in discovering a site among the Mourne Mountains for a private sanatorium in a secluded valley three miles north-west of Rostrevor. The original house I utilise for my own dwelling-house, and have built two blocks of bed and bath rooms and several small huts or bungalows of various simple designs.

The special feature in all my bedroom windows is that the sashes lift out altogether instead of being hinged either at one side or in the middle,

Digitized by Google

In winter, before each patient goes to bed, an attendant places the sash in its frame, while he is dressing and undressing, removing it again immediately afterwards.

Cubic area of the room itself is, when one has thus one side of the room removed, a matter of comparatively small moment. In the Forster Green Hospital some of my patients have their beds right out on the verandah, and they certainly do quite as well there as in ordinary bedrooms.

M. le Dr. F. MORIN (*Leysin*): Le traitement de la tuberculose pulmonaire par la méthode du sanatorium a maintenant fait ses preuves. Tous les pays l'ont adopté. M. le Professeur Brouardel, dans le magistral rapport qu'il a présenté hier aux membres du Congrès, a énuméré les efforts qui sont faits actuellement un peu partout pour lutter contre la tuberculose. Je lui demande humblement la permission d'ajouter la Suisse à la liste des pays qui travaillent énergiquement dans ce sens. A côté des grands sanatoriums de Davos, d'Arosa, et de Leysin, qui sont surtout destinés aux malades de la classe aisée, la Suisse possède ou construit actuellement un nombre assez grand de sanatoriums populaires destinés aux malades de la classe ouvrière. Je citerai ceux du canton de Bâle, à Davos; de Wald, de Braunwald, d'Heiligenschwendi, de Leysin, où un sanatorium de quatre-vingts lits va remplacer un asile de trente lits en exploitation depuis plusieurs années.

Mais il reste bien des points difficiles dans l'entreprise colossale qui consiste à généraliser cette méthode du traitement par le sanatorium.

Permettez-moi d'en aborder brièvement un ou deux devant vous.

1. Et d'abord,—*Où placer le sanatorium?* Cette question est fort importante, et doit recevoir une solution différente suivant les pays et suivant les catégories de malades à soigner.

Dans les pays où cela est possible, et pour tous les cas qui ne présentent pas une contre-indication spéciale, je n'hésite pas à répondre que le climat d'altitude est le meilleur, et que le maximum des chances d'amélioration est fourni par le traitement combiné du sanatorium et du climat d'altitude.

Les statistiques que j'ai pu établir à Leysin, et qui portent sur 500 malades soignés au sanatorium, indiquent des résultats très favorables.

Ainsi pour les maladies du :—

	Guér.	Am.	Totaux des résultats favorables :
1er degré	50 pour cent	... ... 40 pour cent	... ... 90 pour cent.
2e ,, ...	10 ,,	... ... 67 ,,	... ... 77 ,,
3e ,, ...	3·2 ,,	... ... 48 ,,	... ... 51 ,,

Cependant, il est évident que l'on ne peut pas envoyer tous les malades dans un sanatorium d'altitude.

Il faudra pour la grande masse de ces malades, en particulier pour ceux de la classe ouvrière, bâtir des sanatoriums à la campagne, dans des endroits salubres, bien situés, abrités, ayant un air pur, sec, et qui soient aussi rapprochés que possible des endroits habités par les malades.

2. *Quels malades faut-il admettre au sanatorium?*

Digitized by Google

On dit généralement, et avec raison, qu'il vaut mieux n'admettre que des cas curables. Mais ces cas ne sont pas toujours faciles à reconnaître; le pronostic d'un cas de tuberculose est extrêmement difficile à poser. Tel malade, en apparence légèrement atteint, s'aggravera fatalement, même s'il est placé dans les conditions les meilleures; tandis que tel autre, paraissant irrémédiablement perdu, s'améliorera d'une façon inespérée.

D'autre part, il y a un très grand avantage à hospitaliser les tuberculeux à tous les degrés, et ici l'Angleterre nous donne depuis longtemps un grand exemple. Cela permet d'abord d'isoler le malade et de l'empêcher de continuer à être un danger pour sa famille et ses voisins, ensuite cela le soumet à une éducation salutaire et qui sera fort utile à lui et aux autres lorsqu'il rentrera dans la vie commune; enfin, cela lui donne des chances d'amélioration qu'il n'aurait pas rencontrées dans le milieu où il vivait précédemment.

Il me paraît qu'il y aurait un moyen de résoudre la difficulté en multipliant les hospices de tuberculeux, tout en les organisant comme de vrais sanatoriums. Ces hospices ne seraient pas très éloignés des villes. On y recevrait tous les tuberculeux, et au bout de quelques semaines ou de quelques mois, on ferait un triage en suite duquel les malades en voie d'amélioration seraient dirigés sur le sanatorium proprement dit en pleine campagne, ou sur une altitude si le pays le permet.

3. *Combien de temps faut-il garder le malade au sanatorium?*

On parle habituellement d'un séjour de trois mois. Je crois ce temps insuffisant; et il faut chercher à garder le malade plus longtemps; mais il y a là l'une des plus grosses difficultés du problème à résoudre. M. Brouardel nous a montré hier comment on pourra peut-être y arriver. C'est ici que les sociétés de prévoyance, d'assurance, de secours mutuels, de bienfaisance, peuvent intervenir avec succès en s'occupant soit des malades soit de leurs familles.

Le système du dispensaire, tel qu'il nous a été exposé hier, et tel qu'il a été institué par M. Calmette à Lille, peut rendre de très grands services, soit pour envoyer les malades en temps utile dans les sanatoriums, soit pour soigner médicalement et hygiéniquement ceux qui, pour une raison ou pour une autre, ne peuvent y être admis, soit enfin pour continuer à surveiller et à diriger les malades sortis du sanatorium; car il y a là encore un côté important de l'œuvre qui nous occupe. Le tuberculeux ne doit pas cesser d'être surveillé avant, pendant, et après son séjour dans le sanatorium.

C. K. STONE, M.D. (*Boston, U.S.A.*): Mr. President, this is not intended in any way to be a report of Massachusetts State Sanatorium at Rutland, but simply on behalf of my friend, Dr. V. Y. Bowditch, who was unable to be present, to call attention to the good work being done at this institution. The detailed work may be found in the reports of Drs. Bowditch and Clapp, in the Fourth Annual Report of the Trustees of the Massachussetts State Sanatorium at Rutland, September, 1900.

The State Sanatorium at Rutland was first opened for patients in October, 1898. The site of the institution is in the little town of Rutland,

Digitized by Google

forty to sixty miles from the sea. It is on elevated land, and sheltered by the hills from the storms, while open to the sun. There are extensive grounds about, which are gradually being developed. Flower and vegetable gardens, as working places for the patients, are to be developed. The number of patients which can be provided for is 200, and by the new appropriations the capacity of the institution will be increased to 250, which is considered as the limit of the number of patients that it is desirable to have under one administration. Only incipient cases are taken.

The State of Massachusetts has about 2,000,000 inhabitants; consequently there is no lack of material for the formation of good statistics. During the last year Dr. Bowditch reports 39·7 per cent. of arrested cases; rest, 52 per cent. improved, and 7 per cent. of not improved. The patients are expected to live in the open air at all times, and at night the temperature in the sleeping wards is often 40° F.

The State has been so impressed with the good work, that it has appropriated the sum of $110,000 for the completion of the administration building, laboratory, and enlargements, together with a hall for entertainments, better dining facilities, etc.; and in addition the sum of $60,000 for maintenance expenses during the ensuing year.

Dr. Bowditch specially wished to express his belief that it was, after all, the true way of living which was responsible for the results he has obtained. In his work at a small sanatorium at Sharon, only twelve miles from the sea, he has had practically the same results as at the more inland higher altitude conditions. Of sixty-four cases treated in the past five years, a little more than one-third were discharged as arrested. In two the disease has returned. Two, contrary to advice, returned to their old ways of work and living, and relapsed and died; and two others died, one of *operation*, without any symptom of tuberculosis; the other of rectal disease, cause unknown. Good accounts are received from all the others, the majority of whom have been away from the sanatorium for over two years.

Dr. Bowditch is very strong in his belief that the treatment in sanatoria near our large cities is of the greatest value, and at the same time does not involve the expense of long journeys or the absolute separation from friends. In conclusion, I want to say from my own experience, which has been for the most part with advanced cases, that I most fully concur with Dr. Burton Fanning's closing remarks, that many well-advanced cases, when put under proper sanatorium methods of life, even in the centre of a large city, often get a new lease of life and happiness.

Mr. G. W. HAMBLETON, L.R.C.P.I.: I have listened with great pleasure to the discussion on this important subject. We require many more sanatoria, and when the great advantages such institutions can give are fully realised I feel sure this want will in due course be supplied. But we cannot expect philanthropists and others to place the required number at our disposal until better results have been obtained. The open-air treatment does not do all that can be done for the consumptive patient. We can make a great advance on that. There are

Digitized by Google

means by which, in such institutions, we can secure results never before attained. Importance is generally attached to personal experience. I speak as an ex-consumptive who by such means completely recovered twenty-five years ago, and I have applied them with satisfactory results in upwards of fifty cases. We all, I think, agree that in pulmonary tuberculosis we have to deal with a toxic alkaloid, but we differ as to its source of origin. I am satisfied that it is a natural product, normally present in the system, and that its non-elimination is the cause of the disease. I therefore eliminate that toxine, and so does the open-air system of treatment, but only, if I may so put it, in a passive way, and only to a comparatively small extent. We can eliminate the toxine by increasing the functional activity of the skin, kidneys, and alimentary canal, by baths or sponging, diaphoretics, diuretics, and saline aperients. Whether all or only one or two of these functions are to be increased depends upon the extent of the disease and the state of the patient. In the early stage I have found the prescription of two baths in the twenty-four hours, a stimulant alkaline mixture containing diaphoretics and diuretics, a mild saline in the morning, and later on tonics, amply sufficient. But in advanced cases, according to the circumstances of the case, I have ordered the bath and mixture to be taken every one, two, three, four, or six hours. I need not say that great care must be taken to secure good personal and general hygienic conditions for the patient; that his clothing must be light, warm, and loose, and neither impede the free ventilation of the skin nor restrain the movements of the thorax. His food must be good, and the appetite carefully cultivated, not forced. We tell the patient to spend as much time as possible in passive or active, so soon as he is able, exercise in the open air, but he must not attempt to actively develop the lungs until the disease has been for some time arrested. Then we place the patient under conditions that progressively make increasing demands for the use of the lungs, care being taken to avoid strain or over-exertion, such as slight ascents on the neighbouring hills, deep breathing followed by full expiration, gradually increased in number and depth, and exercises that tend to develop the muscles of the thorax.

Under this system of treatment the chest symptoms are immediately relieved, pain, cough, and expectoration speedily disappear, and the area of breathing, the vital capacity, the chest girth and extent of movement progressively increase. The temperature tends to the normal, the general state improves, weight increases, and there is a feeling of health and strength. Soon, in the early stage, there is complete arrest of the disease, no symptoms, good general health, fair weight, normal temperature, a good breathing capacity, and ultimately complete recovery. I have seen the chest girth increased from two to four and a half inches, the range of movement from two to four inches, and the vital capacity from 100 to 200 cubic inches.

The PRESIDENT (Sir Richard Douglas Powell): Ladies and Gentlemen,—We have listened to-day to a very prolonged and very interesting and instructive discussion. I am extremely sorry that in the endeavour to

Digitized by Google

apportion the speakers to represent all the nationalities and the interests on this subject, it has been impossible to bring in the names of all the speakers one would have wished to have heard. Time passes on regardless of our personal regrets or interests. I hope that some of those speakers will find an opportunity to-morrow for the reading of their papers. The discussion which has taken place to-day has proceeded, I think, fairly on those headlines which I very much hoped it would be proceeded upon. Perfect sanitation has rather been taken for granted as a necessity in the treatment of consumption. Skilled alimentation has been advocated by a large number of the speakers, and we have had much additional light on the advantages of it. Controlled metabolism, the regulations of temperature, and the exercise and the ingoings of these consumptives, so as to deal essentially with the question of feeding, have been largely dwelt upon, with great advantage. The usefulness of sanatoria in the treatment of consumption for, at all events, a large number of cases, has, I think, been generally acquiesced in, and it has even been proposed that we should pass some formal resolution on that subject; but the resolution which will be the outcome of these several meetings in the several sections will come before a general meeting of the Congress on a later occasion. It has therefore been thought that it would be rather out of place for us to pass any definite resolution on any one point now. (Hear, hear.) Again, I am very pleased to have observed that the human interests and recreations of those afflicted with consumption, and who have to be sequestered for a time in these public institutions, have not been unthought of. I think that this is a very important point, and I am very glad to have seen it has not been neglected. There have been many other points of interest, but at this late hour I do not propose for a moment to touch upon them. I am only making these few remarks in the absence of Professor Clifford Allbutt, who I thought would perhaps have been here to wind up this discussion. I think that concludes the business of the morning.

Digitized by Google

A PLEA FOR SEASIDE SANATORIA FOR CHILDREN, ESPECIALLY FOR THE PREVENTION AND TREATMENT OF SCROFULOUS COMPLAINTS.

By Sir HERMANN WEBER, M.D. Bonn, F.R.C.P. Lond.,

Consulting Physician to the German Hospital, London, and to the Hospital for Consumption, Ventnor.

In former times the so-called scrofulous diseases were much discussed by the medical profession and the public, but comparatively little attention is paid to them at present, while the more fatal pulmonary tuberculosis commands general attention. Yet there is good reason why scrofulous affections should in an equal measure engage our sympathy and attention, firstly, since the majority of them are likewise of tubercular nature and intimately allied to other forms of tuberculosis, and often lead to pulmonary or to acute meningeal tuberculosis; and secondly, on account of their causing infinite suffering and misery to the patient and often causing death or invalidism or a crippled state for life. By treating scrofulous affections we therefore not only endeavour to cure the existing diseases, but also to prevent pulmonary tuberculosis.

The principal means of cure are:

Life in the open air;

Good food adapted in quality and quantity to the patient's condition;

Hydrotherapeutic measures, such as bathing in the open sea or in tepid sea water, friction of the body with cold water, etc.;

Active or passive exercise or rest in the open air, regulated according to the nature of the affection.

Surgical treatment is required in some cases, and is more successful in the pure air of the seaside than in inland towns; but the majority of diseases of bones and joints, of enlargements of glands and scrofulous ulcers, heal without active operative interference, though bandages are often indispensable.

Such treatment cannot be carried out at the homes of the children of the poor, and frequently even not at the hospitals of large towns; but in the pure air of the seaside (and also of inland places, especially at high elevations) the treatment, if commenced early, almost always results in complete cure.

Those who are rich can make the most necessary arrangements in private houses or establishments, but the children of the poor have no chance excepting in public sanatoria; and for the great majority of them sanatoria at the seaside are preferable to those inland, on account of the more vivifying effect of the sea air, which undergoes constant changes by the regular

Digitized by Google

local currents of air and by the stronger general winds, and on account of the facility of bathing in the open sea or in tepid sea water. Experience shows that at the seaside the energy of the nervous system, the appetite and digestion, and the whole organism are more rapidly improved than at inland localities. Inland sanatoria, however, can likewise be rendered most useful, and are, indeed, preferable in cases of scrofulosis complicated already with pulmonary tuberculosis, or in cases of pulmonary tuberculosis alone, since most of them bear high winds badly. Localities situated at high elevations, as for instance Samaden, exercise a very beneficial influence, not only on pulmonary but also on glandular affections, and on the so-called surgical tuberculosis of joints and bones. This remark is, however, not to be understood as if we did not recognise that great results can be obtained at well situated and well managed sanatoria in lower inland regions. Great and welcome lessons, for instance, are to be learnt from the benefit effected by the "Œuvre d'Ormesson," at Ormesson and Villiers-sur-Marne, near Paris.

As the subject is so very large, we will confine ourselves at present to the sea-side sanatoria for children, as means of prevention as well as cure. Although we must attend to diseases already developed, we must, if possible, not wait till the scrofulous or tuberculous disease is already fully established, but must begin our treatment at the first indications, when we can gain in two months what in advanced cases barely can be effected in two years.

The poor children who are most benefited, and who specially require the sanatorium treatment, are those affected with :—

1. General weakness and deficient nutrition.
2. Tardy and imperfect recovery from various acute diseases such as measles, whooping cough, scarlet fever, diphtheria, influenza, etc.
3. Anæmia.
4. Rachitis.
5. Scrofulous and tuberculous swelling of the lymphatic glands.
6. Scrofulous and tuberculous inflammation of the joints, including hip-joint disease and Pott's disease.
7. Adenoid affections of the throat and nose.
8. Scrofulous ophthalmia.
9. Scrofulous skin diseases.

For the treatment of the majority of the cases it is not sufficient to use any ordinary house, but we require a properly arranged sanatorium which must possess large rooms with abundance of light and air, with windows from the ceiling to the ground, through which the patients' beds can be moved on the adjacent balconies or terraces; the patients who cannot walk must lie there from morning to night, and many with advantage also, with proper shelter, during the night in fairly good weather. The rooms for meals and games must likewise be large and airy. Pavilions of moderate size, of one or two storeys only, are preferable to large blocks having two or three storeys. A pavilion for isolation of infectious cases is a necessity, and a well-arranged

Digitized by Google

operating room is likewise indispensable, although in most cases, if taken early, the necessity of operations can be avoided. Boys and girls ought to be in separate pavilions when older than five years, but may be in the same rooms up to that age.

There ought to be on the sea-shore sheds open on two sides, with rotatory arrangement, for shelter against wind and rain, and to some degree also against too fierce a sun. These shelters are especially necessary and must be numerous, if the balconies for lying out are inadequate, or too far away from the actual shore. To these shelters the little patients who cannot walk must be carried on comfortable kinds of bed-stretchers, and remain there during the whole day.

There must be good arrangements for bathing in the open sea and for tepid sea-water baths, to be used in winter as well as in summer. The resident medical officer must prescribe the diet for every single case, as also the nature and amount of walking and playing, and of Swedish or other gymnastic manipulations, particularly for spine or joint cases.

The beneficial effect of seaside sanatoria can be greatly increased by providing each sanatorium with a *hospital* or *sanatorium boat*, arranged for the accommodation of the sick children, on which, in suitable weather, the invalids can be taken out to the open sea for shorter or longer periods of the day, since it is indisputable that the air on the high sea itself has a more powerful effect on the organism than that on the shore.

The duration of the stay of patients at the sanatorium ought not to be fixed by fast rules, but must depend on the nature of the case, and be left entirely to the judgment of the medical attendant. It may vary from a few weeks to a few years. To limit it to two or three months by the rules of the institution is absolutely wrong. If the patients are admitted at the beginning of the disease or at the first signs of the threatening, a short term will mostly suffice; while advanced cases of hip-joint disease or Pott's disease may require years, and ought not to be discharged before the cure is as complete as the circumstances admit.

Another mistake which is made at many sanatoria is the restriction of the treatment to the warmer months of the year. Although the weather at the northern sea resorts is not always agreeable during winter and spring, the patients at seaside sanatoria are under infinitely better influences than in their homes, or even in ordinary hospitals or infirmaries in crowded towns. The patients sent home during winter lose there often more than they had gained at the sanatorium during summer.

The number of children who require sanatorium treatment at the seaside is very large, and the number of beds available for them in England is very small. There are perhaps three hundred beds in the seaside sanatoria of England, while five thousand would certainly not be too many. Every hospital for sick children ought to have a seaside branch. It is almost incredible that there should be this strange defect in the hospital accommodation of England, especially when we reflect that the whole of the United Kingdom has such splendid sea-coasts, and when we further consider that

Digitized by Google

England was the first country to awake to the great importance of this matter by establishing, on the suggestion of Dr. Lettsom and Dr. Latham, the General Sea-bathing Infirmary at Margate in 1791. The next country to move was Italy, which half a century later founded a sanatorium at Viareggio (1841); and France followed in 1847 by the sanatorium at Cette. While France has made great progress, and deserves the highest praise for her enlightened philanthropy, England has remained almost stationary in this matter. The town of Paris alone maintains in its sanatoria at Berck-sur-Mer about 1,034 beds for scrofulous and rachitic children all the year round, and has also many beds in several other seaside sanatoria on the French coasts. At Berck-sur-Mer, in addition, the benevolent Rothschild family gives perfectly gratuitous treatment to one hundred children in the beautiful Hôpital Rothschild, likewise, summer and winter. France has numerous other seaside sanatoria along her different coasts, mostly under the direction of the Assistance Maritime des Enfans Scrofuleux et Rhachitiques and l'Œuvre des Hôpitaux Marins. Independently of these sea-side sanatoria France possesses, as already mentioned, the admirable inland sanatoria for tuberculous children at Ormesson and Villiers-sur-Marne (Œuvre d'Ormesson).

How is it that England, with its wonderful sea-coasts, and with its many excellent institutions for the sick, erected and maintained by private gifts, neglects this important matter? We cannot think otherwise than that the crying need for help and the possibility of bringing this help, are unknown to the philanthropists of this country, men as well as women. If they were aware of the great sufferings which the poor children affected with Pott's disease of the spine, with hip-joint disease, with caries of bones, or with tuberculous inflammation of joints, undergo in their wretched homes, and how large a proportion of them become consumptive later on, or cripples for life, or die after protracted misery; and if they knew, at the same time, that the majority of them can be cured entirely, and that many of the others can be restored to such a condition that they can earn their bread and can enjoy life, provided they are sent early enough to sea-side sanatoria, many philanthropic persons would surely assist in supplying means to found numerous such establishments. It may be difficult to obtain all the means required by private benevolence; but the subject urgently demands also the attention of public bodies, such as the County Councils and the Poor Law organisations. Associations like the Trade Unions ought likewise to feel the obligation to contribute their share towards the cure of the sick children of their members. Universal compulsory assurance of the working classes would also bring important help. The subject is so important and so large that I venture to suggest that an "*Association for the Erection of Seaside Sanatoria*" should be formed, as a sub-division of the *National Association for the Prevention of Consumption and other Forms of Tuberculosis.*

Digitized by Google

THE CLASSIFIED POPULAR SANATORIUMS.

DR. G. KUSS,

Physician in Chief of the Sanatorium of Angicourt.

ABSTRACT.

AMONG the reasons which prevent obtaining the maximum of useful results in the popular sanatoriums, there is one which must occupy the first place: it is, for any given sanatorium, the uniformity of the conditions of its operating. This uniformity contrasts strangely with the different varieties of tuberculosis treated in the popular sanatoriums.

The first category of patients includes the frail and delicate persons who have need of prolonged rest and whom a genuine sanatorium cure can give back to active life; it is impossible to treat those successfully elsewhere than in an institution possessing a sufficient material installation and an uninterrupted careful medical direction.

A second category is formed by the resistant patients who are continually without fever (*apyrétiques*), with fibrosing inactive lesions; these are real tuberculous patients, spitting bacilli, but whose morbid evolution stops and retrogrades as soon as they are put into the country with a relative rest, in good hygienic conditions. What suits them best is an "anyhow made-up sanatorium" (*sanatorium de fortune*), worked out in the conditions of greatest economy, under the intermittent supervision of a doctor of the vicinity, who should come two or three times per week.

A third category is formed by the patients who have abnormal physical signs, especially of cicatrisation, in whom signs of failing health excite a fear of the return of the tuberculous process, without the possibility to state at the present moment if it is to be ascribed to the tubercles or not. It is enough to procure for these patients, only suspected and not contagious, an air cure with half their liberty, in the country, in a convalescent home, to which an agricultural colony could be adjoined.

These three forms of different asylums for tuberculous patients who may be healed, exist and work at present, but in bad conditions, because the patients they receive are not adapted to the resources they possess. To keep, in a genuine sanatorium, really worthy of the name, patients for whom these ideal conditions are superfluous, is a real waste of money; the inconveniences become more serious when all kinds of tuberculous persons are packed together in an institution, too thoughtlessly baptised with the name of sanatorium! This *sanatorium de fortune* is liable to become a sanatorium of infection and misfortune.

Therefore it is necessary that a particular selection should precede their classification.

Is this selection possible by a consultation made in the city? Certainly not.

In order to be fixed as to the condition of these patients, it is absolutely

Digitized by Google

necessary to have an eye upon them for some time in the service of a hospital. This service must be a special service, provided with all the resources of treatment required by the hygienical cure; provided also with all the means of investigation which help the hesitating clinical diagnosis. It ought to be managed by a specialist having enough time to make these long and circumstantial studies of the patients; and, finally, it must be located in the country, in a pure air, in order to judge under the best circumstances of the resources of vitality possessed by the patients. This experiment of the country is in itself more important than a great many other clinical explorations, but it is evident that it should not be far from town, so that the travelling expenses may be small.

The sanatorium of selection thus defined would have several functions.

Receiving from the city patients supposed curable, it would pass them, so to say, through the sieve; after a long enough examination it would send back (to entrust them either to an asylum for consumptive people or to a dispensary) the tuberculous patients not offering the guarantees of curableness which they seemed to offer at first sight; it would also eliminate the patients who are decidedly false tuberculous patients. The remaining patients should be divided into several groups, according to the nature of the disease; the frail and delicate should remain at the sanatorium which, possessing the medical staff and all the necessary installations, could offer them the best chances for being cured; the others would be sent to a *sanatorium de fortune* or else to a convalescent home.

A group of popular sanatoriums and of annexed institutions for a great city's tuberculous patients who can be cured, organised in the above-mentioned manner, could with the minimum of mistakes and the minimum of expense, oppose to each form of tuberculosis that can be healed, the most suitable manner of hospitalisation, and, on the other hand, prevent at the sanatoriums the residence of the numerous patients for whom, unfortunately, the hygienic dietetic cure cannot have any useful result. Finally, the going through the sanatorium of selection of lots of patients who stay there only a short time would have the valuable advantage to teach to all those tuberculous patients the indispensable cautions to take, and which they need so much to know. Considered from this point of view, nothing could take the place of the "object-lesson" given by a well-working sanatorium.

LES SANATORIUMS POPULAIRES DE RÉPARTITION.

PAR M. LE DR. G. KUSS, *Médecin en chef du sanatorium d'Angicourt.*

PARMI les raisons qui empêchent d'obtenir dans les sanatoriums populaires le maximum d'effets utiles, il en est une qu'il faut mettre au premier plan: c'est, pour un sanatorium donné, *l'uniformité* des conditions de fonctionnement, uniformité qui contraste singulièrement avec la *diversité* des formes de tuberculose qui sont justifiables du sanatorium populaire. Alors même que le

Digitized by Google

choix des malades est fait, à la consultation de la Ville, avec un soin jaloux, il n'en est pas moins certain que, parmi ces tuberculeux sélectionnés, tous présumés curables, des différences fondamentales sont créées par l'état variable des lésions, la résistance plus ou moins grande du cœur, la sensibilité individuelle aux causes de fièvre, les aptitudes réactionnelles du terrain, etc. On est amené tout naturellement à *catégoriser* ces malades.

Les uns, sujets fragiles et délicats (à petites ou grosses lésions, peu importe), ont besoin d'un repos prolongé, souvent même du repos au lit, d'une suralimentation soigneusement dosée et prudemment conduite, d'une surveillance médicale incessante et attentive ; ce sont de *vrais malades de sanatorium*, impossibles à soigner efficacement ailleurs que dans un établissement fermé possédant une installation matérielle suffisante et une direction médicale continue.

Une deuxième catégorie est constituée par des malades résistants et constamment apyrétiques, à lésions sclérosantes torpides dont l'évolution s'arrête et rétrograde sous l'influence du repos relatif dans de bonnes conditions d'hygiène. Ces tuberculoses bénignes, qui spontanément tendent vers la guérison apparente, fournissent de magnifiques succès aux statistiques des sanatoriums, mais elles fournissent des succès non moins beaux aux médecins qui, dans leur clientèle privée, ont envoyée de tels malades se mettre au vert à la campagne dans des conditions d'installation quelconque. Seulement, comme ces malades sont de vrais tuberculeux à lésions généralement ouvertes, on ne peut songer à les soustraire à toute surveillance ; dans leur intérêt, comme dans l'intérêt des localités où ils séjourneront, il faut les grouper dans des établissements où ils ne sont pas livrés à eux-mêmes, où les imprudences leur sont interdites, où une surveillance médicale *intermittente*, mais réelle, peut s'exercer sur eux. Un "*sanatorium de fortune*" visité deux ou trois fois par semaine par un médecin d'une localité voisine remplira ces indications.

Reste enfin une troisième classe de malades, fort intéressants ceux-là, mais dont il est vraiment très difficile d'apprécier l'état de santé exact. Je veux parler des sujets porteurs de signes d'auscultation surtout cicatriciels, qui subissent, sous une influence quelconque, une déchéance de l'état général. Comme l'exploration de la poitrine ne révèle pas de lésion tuberculeuse en évolution actuelle, que l'expectoration, quand elle existe, est peu marquée et privée de bacilles, on est très embarrassé pour savoir si ces malades sont ou non de vrais tuberculeux. Comment décider si derrière ces zones de sclérose ne se cachent pas des foyers caséeux capables d'influencer la santé ; une étude détaillée du malade, certaines épreuves thérapeutiques permettent de serrer le problème de plus près, mais ce sont des recherches longues et délicates. Quand on reste dans le doute, il est absolument indiqué de soigner ces malades comme de vrais tuberculeux et de leur assurer le bénéfice du traitement hygiénique ; mais, pas plus que pour les sujets de la catégorie précédente, la cure de sanatorium n'est indispensable ; c'est le repos dans un air pur et tonique qu'il faut à ces malades et, comme ils n'ont pas de lésions ouvertes, que ce sont simplement des suspects, fatigués, anémiés, déprimés, neurasthénisés, on peut se borner à leur procurer une cure d'air en semi-liberté

Digitized by Google

à la campagne, dans une *maison de convalescence* à laquelle on pourrait annexer une colonie agricole.

En résumé, *à ces trois catégories de tuberculeux curables conviennent des formations hospitalières différentes:* aux malades fragiles et délicats, prompts aux poussées bacillaires, le *vrai sanatorium* avec direction médicale continue; aux formes torpides et résistantes de tuberculose apyrétique le *sanatorium de fortune* avec surveillance médicale intermittente; aux suspects, la *maison de convalescence.*

A l'heure actuelle, ces trois formes différentes d'asiles pour tuberculeux *existent et fonctionnent*, et il me serait facile de citer des noms précis; mais ils fonctionnent dans de mauvaises conditions, parce que les malades qu'ils reçoivent ne sont nullement adaptés aux ressources qu'ils possèdent.

Conserver dans un vrai sanatorium digne de ce nom des malades pour lesquels ces conditions idéales sont parfaitement superflues, c'est un véritable gaspillage. Les inconvénients sont plus sérieux quand on entasse toutes sortes de tuberculeux dans des établissements baptisés à la légère "sanatoriums"! on s'expose à ce que le sanatorium de fortune devienne alors un sanatorium d'infortune et d'infection.

Il faut donc qu'un choix minutieux des malades précède leur répartition; tâche importante entre toutes, puisque les erreurs de pronostic se traduiront nécessairement par un déficit budgétaire et par une mauvaise utilisation des instruments de combat dont on dispose.

Peut-on décider la répartition des malades dans une consultation faite à la ville?

Certainement non: il est déjà fort difficile de choisir, dans ces conditions, les tuberculeux qu'on doit accepter au sanatorium et ceux qu'on doit refuser. Dans la plupart des cas, l'auscultation, à elle seule, ne suffit nullement pour établir un pronostic; on est forcé d'y joindre l'examen complet du malade, le facies, l'état du pouls, la résistance aux causes de fièvre, l'état des fonctions digestives, etc., et tous ces éléments d'information peuvent être profondément viciés dans leur signification par les conditions de fatigue, de surmenage, de misère et d'angoisse morale où se trouvent les malades. On est sans cesse exposé à commettre des erreurs dans un sens ou dans l'autre; à plus forte raison ne saurait-on espérer *classer* d'une manière exacte les sujets que l'on estime pouvoir guérir.

Pour se faire une opinion sur ces malades, il faut, de toute nécessité, les avoir suivis quelque temps dans un *service hospitalier;* ce service doit être un service *spécial*, pourvu de toutes les ressources de traitement exigées pour la *cure hygiénique*, pourvu aussi de tous les *moyens d'investigation* qui permettent de venir en aide au diagnostic clinique hésitant: il doit être dirigé par un médecin *spécialisé* qui ait le temps de faire ces études de malades, longues et minutieuses; enfin, il doit être situé *à la campagne*, dans un air pur, pour qu'on puisse juger, dans les meilleures conditions, des chances de guérison que possèdent les malades: chez beaucoup de tuberculeux qui arrivent au sanatorium pâles, défaits, anorexiques, languissants, subfébriles, deux ou trois semaines de repos au grand air suffisent pour déterminer une transformation radicale: à vue d'œil les malades se colorent et engraissent, leur appétit se

Digitized by Google

réveille, la température redevient normale . . . et, ce qui montre bien l'influence toute spéciale des conditions climatériques, c'est que ces transformations s'observent aussi chez des sujets qui sortent d'un hôpital ou d'un sanatorium urbains. Inversement, d'autres malades restent atones, déprimés, mangent mal, n'engraissent pas. Si l'on cherche la raison d'être de ce manque de stimulation, on la trouve, bien souvent, dans une affection chronique des voies digestives, dans un état neurasthénique ancien, ou dans des signes plus directs qui montrent que l'organisme est touché profondément par le processus tuberculeux. Aussi cette "épreuve de la campagne" est, à elle seule, plus importante que beaucoup d'autres explorations cliniques; elle contribue à faire connaître la puissance de vitalité du malade. Donc, le service en question sera situé en pleine campagne, mais assez près de la ville pour que les frais de transport des malades demeurent très restreints.

Au service hospitalier ainsi défini convient le nom de *sanatorium de répartition.* Ses fonctions seraient multiples. Recevant le lot des malades présumés curables envoyés de la ville, il le passerait au crible, pour ainsi dire.

1°. Après examen suffisamment prolongé, il renverrait (pour les confier soit à un asile de phtisiques, soit à un dispensaire) les tuberculeux qui ne présenteraient pas les garanties de curabilité qu'on avait cru constater à première vue. Cette élimination, si pénible soit-elle, est nécessaire dans un sanatorium populaire, où l'on doit pouvoir obtenir en cinq ou six mois des améliorations *durables;* or, le pronostic de la tuberculose est entouré de telles difficultés que les médecins les plus compétents ne peuvent pas prévoir, à coup sûr, le résultat que donnera la cure; c'est l'expérience faite au sanatorium sur un malade donné, qui doit trancher la question. Remarquons ici, en passant, que les malades qui sont justiciables du sanatorium populaire appartiennent à deux groupes différents:

Les uns sont des tuberculeux *tout à fait au début,* mais, dans la classe pauvre, ils sont rarement dépistés à temps; de plus, ils constituent bien souvent de mauvais malades, car on ne sait jamais, quand une tuberculose commence, comment elle évoluera, et les malades qui arrivent avec une lésion en germination et des illusions d'avenir qui paraissaient légitimes, peuvent très bien avoir une de ces tuberculoses graves, à marche continue, pour lesquelles on ne peut guère espérer un bon résultat, tout au moins dans les conditions des sanatoriums populaires, où la lutte ne peut être prolongée indéfiniment.

Un deuxième groupe est formé par les malades qui, *frappés depuis longtemps par la tuberculose, ont déjà fait leurs preuves de résistance.* Il y a de nombreuses tuberculoses semi-bénignes qui évoluent avec intermittence pendant de longues années; le plus souvent, elles commencent dans l'enfance, restent latentes ou larvées très longtemps, en donnant par intervalles des épisodes aigus: hémoptysie, bronchite, fièvre éphémère, pleurésie, etc. Il y a ainsi une série de feux de paille qui s'allument et s'éteignent vite, jusqu'au jour très lointain, parfois, de l'embrasement terminal qui consume l'individu. Le rôle du sanatorium populaire est précisément d'intervenir au moment d'une de ces poussées éphémères pour éteindre cette poussée et

Digitized by Google

sauvegarder l'avenir. Assez souvent le rapprochement des épisodes aigus et l'aggravation qui en résulte détermine la formation d'une *phtisie fibreuse torpide* compatible, pendant des années, avec une vie active.

Là encore le sanatorium a un rôle très utile (utile à la fois au point de vue humanitaire et au point de vue social), en rendant à ces malades plusieurs années de santé suffisante ; un bénéfice réel et durable peut être obtenu pour eux si l'on a soin d'éliminer les sujets que l'étendue des lésions cicatricielles antérieures a convertis en véritables infirmes.

C'est principalement pour ces malades à tuberculose fibreuse ancienne, compliquée d'une poussée récente, que l'*épreuve préliminaire de la cure de sanatorium* est utile. Il n'est pas rare, au sanatorium, de constater chez de tels malades, qui à l'entrée paraissaient en piteux état, des améliorations profondes extraordinairement rapides, et d'obtenir en quelques mois un bénéfice considérable qui contraste avec l'inutilité des tentatives thérapeutiques poursuivies pendant les années précédentes. Il faut donc se montrer assez large dans les admissions, sous la réserve de renvoyer les malades du même groupe dont l'amélioration se dessinera trop lentement. Le fonctionnement du sanatorium de répartition remplira précisément toutes ces indications.

2°. C'est également au sanatorium de répartition que seraient éliminés les malades qui, décidément, seraient de faux tuberculeux. Pour un certain nombre de ces malades, l'auscultation répétée, l'étude minutieuse de la température après la marche, l'épreuve de l'iodure, la recherche des autres tares organiques pouvant expliquer la mauvaise santé, le chimisme respiratoire, sans doute aussi, et la radioscopie permettront d'écarter le diagnostic de tuberculose. Du reste, il sera toujours prudent, en rendant ces malades à leur vie habituelle, de les faire suivre de près par les médecins d'un dispensaire pour tuberculeux.

3°. Les malades restants seraient, suivant leur forme de tuberculose, divisés en plusieurs groupes : les sujets fragiles seraient conservés au sanatorium de répartition qui, étant en possession du personnel médical et de l'installation nécessaires, leur offrirait les meilleures chances de guérison ; les autres seraient dirigés sur des sanatoriums de fortune ou sur des maisons de convalescence.

En organisant de cette manière un *groupe de sanatoriums* populaires et d'établissements annexes destinés aux tuberculeux curables d'une grande ville, on pourrait, avec le minimum d'erreurs et le minimum de dépenses, opposer, à chaque forme curable de tuberculose, le mode d'hospitalisation qui lui convient et, d'autre part, éviter le séjour aux sanatoriums de nombreux malades pour lesquels, malheureusement, la cure diététo-hygiénique ne permet d'arriver à aucun résultat utile. Enfin le passage au sanatorium de répartition d'une série de malades qui n'y resteraient que peu de temps, aurait l'inappréciable avantage d'enseigner à tous ces tuberculeux les précautions indispensables qu'ils ont tant besoin de connaître. Rien ne saurait remplacer, à ce point de vue, la leçon de choses donnée par un sanatorium fonctionnant bien.

Digitized by Google

CONCLUSIONS.

1°. En raison de la diversité des formes de tuberculose justifiables du sanatorium populaire, on est amené tout naturellement à *catégoriser les malades.*

2°. Pour arriver avec le minimum de dépenses aux maximum de résultats, on doit réserver à chacune de ces catégories une *formation hospitalière différente:* aux malades fragiles et délicats convient un vrai sanatorium digne de ce nom, avec surveillance médicale continue; aux tuberculeux résistants et apyrétiques à lésions sclérosantes torpides, le sanatorium de fortune, fonctionnant très économiquement, avec surveillance médicale intermittente; aux suspects et aux convalescents, une maison de convalescence à la campagne.

3°. La *répartition* méthodique et rationnelle des malades doit être décidée dans un sanatorium bien installé, situé à proximité de la ville; ce sanatorium, recevant le lot des malades présumés curables envoyés de la ville, le passerait au crible pour ainsi dire; il arrêterait au passage les sujets pour lesquels la cure de sanatorium ne se montrerait pas profitable et il répartirait les autres malades dans les établissements dont les ressources seraient adaptées à leur forme de maladie.

TRAITEMENT DES TUBERCULEUX PAR LA CURE D'AIR À L'HÔPITAL PASTEUR DU HAVRE.

PAR M. LE DR. FROTTIER, *le Havre.*

L'ISOLEMENT des tuberculeux ayant été réalisé—à la demande du corps médical—dans les hôpitaux du Havre, ces malades ont été réunis dans divers pavillons de l'hôpital Pasteur, et j'ai obtenu de l'administration l'installation d'une galerie de cure annexée à l'un de ces pavillons, pour le traitement des malades les plus curables.

Ce service fonctionne maintenant depuis le 1er février 1900. Ce qui le distingue particulièrement, et ce pourquoi il m'a paru intéressant d'en parler ici, c'est que, tout en faisant partie d'un hôpital urbain, il occupe une situation vraiment exceptionnelle; bâti dans un parc magnifique, très bien abrité, et situé à une altitude de 70 mètres au-dessus du niveau de la mer, il est, autant que possible, à l'abri des contaminations urbaines.

Si j'ajoute que l'administration m'a consenti, au point de vue du régime alimentaire, toutes les modifications compatibles avec les nécessités budgétaires, il semblera donc que, au point de vue de l'organisation matérielle, ce service dispose de tout le nécessaire pour recueillir de bons résultats dans ce véritable sanatorium urbain.

De fait, après dix-huit mois d'expérience, je demeure convaincu qu'aucune autre méthode que la cure d'air, de repos et d'alimentation, n'en aurait procuré de semblables.

Mais ils sont encore insuffisants à mon avis; et comme les causes de cette insuffisance ne sont imputables ni à l'organisation ni à la méthode, mais aux

Digitized by Google

malades eux-mêmes, comme aussi il est à supposer qu'elles ne sont pas toutes particulières à la population Havraise et doivent se retrouver un peu partout, il importe, je crois, de les signaler afin de s'efforcer d'y remédier dans l'avenir.

Du 1er février 1900 au 1er juin 1901, 112 observations ont été recueillies concernant 105 malades :—

19	ont été	très améliorés,	soit	17 pour cent.
39	,,	améliorés	,,	35 ,,
30	,,	stationnaires	,,	27 ,,
10	,,	aggravés	,,	9 ,,
14	,,	décédés	,,	13 ,,

Sur ces 112 malades, 25 n'avaient pas de bacilles à leur entrée ; tous les autres en avaient et présentaient des symptômes de fonte pulmonaire plus ou moins avancée et plus ou moins généralisée. Tous avaient un mauvais état général, certains n'ont pu quitter leur lit pour faire la cure, plusieurs ont dû le garder un certain temps avant de se lever.

Soixante-douze malades ont gagné du poids, un peu plus de 6 livres en moyenne (de 1 au minimum à 18 au maximum) ; 12 ont restés stationnaires ; 28 en ont perdu.

La durée moyenne du séjour a été de 59 jours pour l'ensemble des malades ; mais il est important de noter qu'elle fut de :—

90	jours	pour	les très améliorés.
54	,,	,,	améliorés.
28	,,	,,	stationnaires.

Les débuts furent assez difficiles ; j'eus quelque peine à imposer aux malades le séjour à l'air en hiver, le repos sur les chaises-longues et surtout l'interdiction de cracher partout.

Mais les véritables obstacles sont ceux qui se déduisent de la courte statistique que je viens de citer, et ce sont ceux auxquels on devait tout naturellement s'attendre :—

1° Les malades viennent se faire soigner trop tard ;

2° Ils ne consentent généralement pas à se soigner assez longtemps.

Or, à quelle catégorie de malades avons-nous à faire ? Pour la plupart à des journaliers des grandes usines ou des quais, ou à des matelots et chauffeurs. C'est par exception qu'ils ne font pas un usage habituel de l'alcool, presque tous en prennent : "Ah ! sans faire jamais d'excès !" On sait chez nous ce que cela veut dire et je ne les questionne même plus sur ce point. Aussi tous sont déprimés ; il est rare que leur appétit habituel, avant de tomber malades, ait été bon, leur résistance est considérablement affaiblie.

Il leur faudrait donc faire à la cure un long séjour pour pouvoir espérer quelque bénéfice durable, alors qu'on a bien de la peine à les retenir, malgré tous les discours et toutes les bonnes raisons.

Quant à les décider à se soigner de bonne heure, c'est fort difficile : "On se soigne quand on ne peut plus travailler."

Telles sont les causes de l'insuffisance de nos succès, et tant qu'elles per-

Digitized by Google

sisteront, nous ne pourrons guère espérer mieux du sanatorium populaire : voilà ce que je me proposais de dire à ceux qui, comme nous, se préoccupent du traitement des tuberculeux pauvres par le sanatorium de quelque manière qu'on le comprenne.

Il faudra envisager ces difficultés pour que la magnifique campagne, entreprise maintenant dans toute la France, porte un jour tous ses fruits.

Mais on ne saurait aussi trop répéter que l'avenir—dans la lutte sociale contre la tuberculose—appartient surtout à la prophylaxie : il sera toujours plus facile d'empêcher les gens de devenir tuberculeux que de les guérir quand ils seront tombés malades ; on fera toujours plus de bien en diffusant les notions de l'hygiène comme se le proposent déjà ces admirables institutions qu'on appelle la " Ligue contre la tuberculose " du Docteur Armaingaud, la Société de préservation contre la tuberculose par l'éducation populaire, pour ne citer que celles-là, en luttant contre les progrès de l'alcoolisme, en réalisant l'assainissement des villes.

Et pourtant, il restera toujours et quand même des tuberculeux auxquels nous devrons nos soins : pour ceux-là la méthode hygiénique représente le seul remède efficace que nous puissions leur offrir, jusqu'au jour où quelque nouvelle découverte—sortie, peut-être, du superbe Institut de la Hauteville—viendra nous donner des armes nouvelles.

Mais je m'empresse d'ajouter, après cette première expérience du sanatorium populaire, que je ne saurais comprendre la cure hygiénique autrement qu'appliquée dans des établissements spéciaux, de vrais sanatoriums. Surtout quand il s'agit de malades pauvres, dont les habitudes anti-hygiéniques sont de règle, c'est une erreur de croire qu'il suffit de leur offrir de l'air, un lit et une chaise-longue, une alimentation suffisante, des conseils du médecin de temps en temps, pour les guérir de leur tuberculose. Les exigences de la cure nécessitent la docilité des malades, la discipline, l'encouragement constant, et par conséquent la présence aussi continue que possible du médecin, qui non seulement les soigne, mais surtout les dirige et les soutient.

Le mot de Sabourn sera toujours d'actualité : " Tant vaut le médecin, tant vaut le sanatorium."

Quant à ces établissements eux-mêmes, la question ne se pose même pas de savoir si l'on doit toujours bâtir ou toujours utiliser ce qu'on trouve à sa disposition. L'exemple que je viens de citer démontre bien qu'on pourra souvent utiliser certains hôpitaux, certaines maisons de convalescence, etc., etc. . . mais, s'il nous faut construire, rien dans la méthode hygiénique ne nous oblige à faire ni grand, ni cher.

Dès maintenant, il me paraît que le premier effort, en vue de l'avenir, doit se porter vers l'éducation du peuple. Il faut que nous fassions pénétrer partout les notions de contagiosité et de curabilité de la phtisie ; il faut que les intéressés deviennent convaincus de l'importance qu'il y a pour eux à se faire soigner de bonne heure et pendant le temps voulu.

Il faut, comme le demande la Commission extra-parlementaire de la tuberculose, qu'on organise les caisses de secours aux familles des malades, les dispensaires ; que les grandes administrations de l'état et des villes

Digitized by Google

donnent les premières l'exemple dans l'organisation de cette campagne au nom de l'hygiène publique.

Il faut ne jamais se décourager, et se souvenir que nos premiers convertis, une fois rétablis, sont nos meilleurs apôtres.

L'ŒUVRE DES SANATORIUMS MÉDICAUX FRANÇAIS.

PAR M. LE DR. VERHAEREN (*Secrétaire-Général de l'Œuvre de la Tuberculose en Algérie; Directeur du Sanatorium d'Alger*).

DANS la guerre à outrance que tous les pays entreprennent contre la tuberculose, la cure par le sanatorium paraît tenir la première place. L'Allemagne a donné l'exemple, la France travaille activement à se mettre à son niveau: depuis quelques années de nombreux sanatoriums y ont été créés, et de plus nombreux encore sont actuellement en voie de formation. Mais il ne suffit pas d'avoir des armes à sa disposition, il faut encore savoir s'en servir; il faut, en ce qui concerne les sanatoriums, que les règles qui doivent présider à leur installation soient bien connues de leurs fondateurs—que leur fonctionnement et leurs indications, les avantages et les inconvénients de chacun d'eux soient connus de tous les médecins; il faut, en un mot, propager le principe de la cure sanatoriale et les ressources que possède chaque pays pour permettre d'y recourir.

C'est le but que nous nous sommes proposé d'atteindre en créant *l'œuvre des Sanatoriums Médicaux Français*, dont j'ai l'honneur de vous entretenir. Ce but est defini par l'article 3 des Statuts, article que voici :—

"L'œuvre a pour but: 1°, d'établir un lien moral et scientifique entre tous les sanatoriums médicaux français; 2°, de vulgariser leurs avantages, leurs procédés, et leurs indications; 3°, de favoriser la création de nouveaux sanatoriums gratuits ou payants, et d'aider ainsi de la façon la plus efficace à la lutte contre la tuberculose."

Et tout d'abord, il convient d'adopter une définition précise du mot "sanatorium"; la suivante est celle que j'ai proposée au Congrès de Médecine Professionnelle de Paris en août 1900, et nous a paru réunir tous les désiderata: "Le sanatorium est un établissement exclusivement médical, destiné aux tuberculeux ou aux tuberculisables, dans lequel sont rigoureusement appliqués par le Médecin Directeur lui-même, ou sous sa surveillance, tous les principes appropriés d'hygiène, de thérapeutique, et de prophylaxie."

Cette définition étant prise comme point de départ, nous avons admis ensuite les trois préceptes suivants, qui en dérivent ou la complètent:—

1°. La cure de la tuberculose par le sanatorium est la seule réellement efficace; tout au moins doit-on dire qu'elle est l'accompagnement obligé de tout autre traitement. D'autre part, grâce à l'isolement relatif et aux mesures d'antisepsie qu'elle comporte, elle constitue le moyen prophylactique le plus sérieux de tous ceux opposés à la tuberculose.

2°. Le sanatorium, pour donner tout repos au malade qui recourt à lui,

Digitized by Google

et par suite au médecin qui l'y envoie, doit être exempt de toute idée commerciale—c'est-à-dire qu'il doit être, ou un établissement de bienfaisance pure et de philanthropie tels que ceux créés par les services d'assistance publique—ou un établissement privé, exclusivement dirigé par un médecin. Le Congrès de médecine professionnelle de Paris en 1900—à la suite d'un rapport déposé par moi sur cette question, a emis un vœu dans ce sens.

3°. "Chaque climat a ses indications bien définies vis-à-vis de la tuberculose pulmonaire, en dehors lesquelles il devient indifférent ou nuisible." Ce principe, qui est une des conclusions de ma communication au Congrès de la Tuberculose de Paris en juillet 1898, est généralement admis aujourd'hui. La France possède actuellement des sanatoriums répondant à toutes les indications—soit construits, soit en construction. Pour la facilité du classement et sans vouloir atténuer les nuances qui les distinguent, nous les avons reportés en trois catégories principales.

1ère Catégorie. Sanatoriums de haute altitude, convenant aux prétuberculeux, aux tuberculeux du 1er degré ne portant ni lésions ouvertes aux poumons, ni complications dans d'autres organes. Le type de ces sanatoriums est celui d'*Aubrac* (*Aveyron*), à 1,400 mètres d'altitude, fondé en 1895 par le Dr. Saunal. Ouvert toute l'année, il consiste actuellement en un chalet de 20 lits—portés à 60 lits par des constructions en cours. Le service médical est assuré par les Drs. de Moncan et Fauvel.

2e Catégorie. Sanatorium d'altitude moyenne convenant aux tuberculoses ouvertes, avec fièvre modérée, aux formes torpides a lésions assez étendues, mais à état général bon.

Sanatorium de Durtol (Puy de Dôme), fondé en 1898 par le Dr. Sabourin, ouvert toute l'année; il comprend environ 30 lits.

Sanatorium de Gelos, complété par celui des Eaux Bonnes (B. Pyr.), fondés et dirigés par le Dr. Portes; le 1er, ouvert d'octobre à mai, contient 15 lits; le 2e, ouvert de mai à octobre, en contient 10. Situé à 800 m. d'altitude, près des sources sulfureuses, ce dernier permet l'application facile de ce traitement à certains malades, à formes torpides, et à élément catarrhal prédominant.

Sanatorium de Gorbio Menton (Alpes Maritimes), fondé en 1900 par le Dr. Malibran.

Sanatorium de Meung sur Loire (Loiret), fondé et dirigé par le Dr. Léon Leriche. Situé a 126 m. d'altitude, il contient 17 lits.

Sanatorium des Pins à Lamothe Beuvron (Loir et Cher), fondé en 1900 par le Dr. Hervé. Son altitude est de 130 m., ses lits sont au nombre de 22.

3e Catégorie. Sanatoriums de climats tempérés chauds, dont la moyenne thermomètrique hivernale est au dessus de 12°. Conviennent aux tuberculeux torpides à lésions étendues, aux formes subaigues, aux porteurs de complications au larynx, à l'intestin, aux reins, etc.

Sanatorium d'Alger, fondé en 1895 par le Dr. Verhaeren, altitude environ 200 m. Il comprend 100 lits, dont 60 réservés aux assistés, dans une section séparée.

Digitized by Google

TREATMENT IN INSTITUTIONS AND IN OPEN-AIR NURSING HOMES.

By E. SYMES THOMPSON, M.D., F.R.C.P.,
Consulting Physician to the Hospital for Consumption, Brompton, London.

The object of this communication is to draw attention to the value of the "Nursing Home," as distinguished from the sanatorium, in the treatment of chest disease. The distinction may not seem at first sight to be great, yet in practice it is found to be considerable. In the Home the patients, being few in number, are under the constant supervision of a highly-trained nurse-matron, and have the daily visit of a skilled medical attendant, able to give ample time to the conduct and management of the case. Whilst the Germans are ready to adjust themselves to the routine life of a great sanatorium, to the insular habits of the Briton the more secluded and private life of the Nursing Home is more conducive to comfort. In conditions of acute disease requiring incessant individual care it is certainly preferable, idiosyncrasies being more readily attended to than in a larger institution. No time is lost in adjusting the treatment to the varying requirements of the case, and in securing that complete rest which has long been recognised in the treatment of tubercular affections of the bones and joints, but which in the treatment of chest disease has not in the past been adequately acted upon. The fact has been recently acknowledged that rest is as successful in arresting the course of tubercular pyrexia as in pneumonia and rheumatic fever. Nature gives us this hint by restricting the movements of the ribs and intercostal muscles, not only in cases of painful pleurisy, but in diseases of the lungs themselves. Dr. A. Ransome has, by means of his two-plane stethometer, demonstrated the degree of limitation of natural movement, but the eye and trained hand alone are sufficient to show that the degree of limitation of movement is often proportionate to the extent and severity of the underlying disease and the constitutional vigour of the patient.

Dr. Aikman, of Guernsey, has also shown more recently that the limitation of respiratory movement in the præcordial area affords a test of the presence of pericardial disease and is often also of prognostic value, because this symptom precedes the development of stethoscopic evidence of inflammatory complications.

As Nature thus demonstrates the importance of rest, we must in our treatment of pulmonary disease take care to secure the required quiescence of the injured organ. We cannot altogether put a stop to respiratory movements, but we can and must limit them. Quiescence can best be obtained by rigid recumbency, and the experience of the open-air treatment shows that a gain in weight, as well as an improvement in appetite and power of assimilation, is thus secured. The blood flows more readily, its quality improves, the lips and cheeks gain colour, and general amendment results.

Digitized by Google

When complete rest is no longer needed, the pyrexia having been removed, the horizontal position should be maintained for an hour after each meal. Thus the feet and knees are less cold than when standing or sitting, and the large quantity of fluid required for the digestive juices can be more readily spared without causing exhaustion. It is during repose that oxygen is most freely absorbed, especially when rest is taken in the open air, the body being protected from chill by means of adequate wraps.

It is curious that in other febrile diseases the patient is more ready to rest in the recumbent position than in consumption, when the patient often desires to continue at work even during the evening febrile exacerbation. It is by firm discipline that the needed control can alone be obtained. This discipline can rarely be secured in the home of the patient.

Whilst rest is often essential, we must be careful not to allow the vital forces of the body, and especially of the muscular system, to run down from disuse; nerves and muscles need exercise, or effete matter will not be eliminated and oxygenation secured.

Dr. Weir-Mitchell obtained for his neurasthenic patients the necessary movement by massage and electricity. In phthisis one can generally attain the desired end by rest after food, and by limiting movement so as to prevent breathlessness, and by keeping the patient in bed or on a couch as long as may be needful without endangering the efficiency of the muscular and nervous system.

When fever has subsided and weight has begun to increase, graduated exercise under skilled direction may be adopted. Exciting games, such as football, cricket, etc., must, of course, be avoided. Whilst fever exists, in however slight a degree, all exercise causing breathlessness must be interdicted. No forced respiration must be allowed.

The blood pressure, the pulse rate, the temperature, and the limitation of respiratory movements must be carefully observed before any increase of active movement can be sanctioned.

Nerve exhaustion, morbid depression, and any exertion which lowers resistance, facilitate the introduction and development of noxious organisms.

In our open-air homes and institutions, as how arranged, catarrhal affections are almost as rare as on the ocean.

The time must be chiefly spent in recumbency or semi-recumbency; long walks being interdicted, exercise must be gentle for months after the apparent restoration of health.

There is no special virtue in the sanatorium or in the Nursing Home which is unobtainable elsewhere, provided that the same authority can be exacted as in a home. Just as a surgeon prefers a surgical home if he has a serious operation to perform, and considers it desirable to remove his patient from his house, so the physician who has a critical case of lung disease has far greater confidence in the result if he can place his patient in a good Nursing Home.

Where the will exists on the part of the patient to carry out to the letter every detail with implicit obedience, much may be hoped for in the patient's own home, but the house must be suitably arranged if we are to secure full benefit. It must be in a dry, airy, healthy position, free from carpets, curtains,

Digitized by Google

and dust. The cooking and dieting must be supervised, and suggestions of kind friends not be accepted if the discipline of doctor and nurse is to be maintained. Even when all these things are attended to, the patient will miss the hopefulness which comes from association with companions who are daily improving and full of confidence.

Every practitioner should have it in his power to carry to a successful issue the treatment of those who are really unable to leave their homes. It must be admitted, however, that even when a wise doctor, a firm and skilled nurse, and an obedient patient are secured, the superior efficacy of the "Home" with a capital letter to the "home" without the capital is as manifest in many cases of phthisis as it is in cases of neurasthenia when the Weir-Mitchell treatment is adopted. In the nervous class of diseases it is almost impossible to obtain good results without removal from home, and in chest disease the difficulty is also great. The thoughtful supervision of every detail of life, of things perhaps small in themselves, makes all the difference between success and failure.

As an illustration of the value of skilled supervision, we may refer for a moment to the report of the Balneological Committee of the Royal Medical and Chirurgical Society, which has prominently placed before the medical profession the value of spa treatment in chronic disease. The spa is valued less for its chemical or medicinal constituents than for the judgment with which the "spa treatment" is applied. It is recognised that the superior value of a spa is dependent more on the skill and discrimination of the medical adviser than on the medicinal waters themselves.

Patients are learning to accept the authority of the spa doctor, to recognise the important function of the trained nurse, and to obey rigid rules of treatment. Whether the treatment is carried on in an "Institution," in a "Nursing Home," or "at home," there is a better prospect of securing obedience than was formerly the case. Doctors and patients alike have learned that by discipline and obedience to rule a cure may be obtained in England as well as abroad. The glamour associated with the foreign spa doctor who assumed the position of king in his own castle is not reached here, still there are many Englishmen who have learned to exercise dominion and to secure obedience.

Whilst the value of climate in treatment is pretty well appreciated, there is a growing recognition of English open-air homes. Judged by results it must be admitted that the progress made in them is exceedingly favourable. The success of the Alpine winter health resorts was largely due to the open air, but even more to the accurate supervision of every detail of life. In the open-air treatment as carried out in foreign institutions, attention to detail has been great. In the English institutions, based on a like principle, the good effects have often been frustrated by the unwillingness of the independent Briton to bow to any authority. German and even French patients are willing to sink their individuality and to submit to well-nigh military discipline, to force themselves to eat piled-up plates of unattractive food, and to spend their time out of doors irrespective of weather, debarred even from the

Digitized by Google

excitement of whist, chess or even dominoes! To the average Englishman or Englishwoman this is a real difficulty—a difficulty, however, that must be overcome. Obedience is the fundamental necessity, pure air, uncontaminated by foul drains, by dust, fur, woollen curtains or carpets, padded chairs or even mats, pictures or bookcases. Above all, air must *never* be breathed a second time.

Whilst on this subject a few words may be added on the mode of access of tubercle. In childhood the most common mode of access is the alimentary canal, the mesenteric glands being first involved. Out-patient practice in our London hospitals, and especially at the Brompton Hospital for Consumption, emphasises this. In adult life it is otherwise. The tonsils and naso-pharyngeal membrane are continually found to be the source by which tubercle enters the system. Laryngeal ulceration is a late, but pharyngeal defect is an early—often the earliest—manifestation of tubercular disease.

By inhalation, the tubercle bacillus is supposed to reach that part of the bronchial tree which will afford a suitable nidus, and this, we are told, is the ultimate bronchiole at the margin of the pulmonary vesicle.

The larger bronchial tubes do not readily become affected. In support of the inhalation hypotheses, physiological experiments with animals infected by being subjected to sprays containing tubercle are quoted; but, on the other hand, it is admitted that it is hardly possible to reach tubercular deposits in the lung by the use of healing or disinfecting sprays, however finely divided. If these regions are outside the reach of sprays it would seem unlikely that the tubercle bacilli would climb so far into the bronchial tree. The true explanation of the inhalation experiments may be found in the fact that the bacilli lodge on the naso-pharyngeal and buccal membranes, and either directly or through the tonsils, or often by deglutition, find their way into the blood stream.

A case giving a coarse example of the mode of infection may be mentioned, in which first tubercular ulceration of the tonsil was noted and treated by lactic acid, scraping, etc. This was followed by enlargement of the cervical glands, and subsequently by pulmonary tuberculosis.

Again, cases of tubercle in bones, joints, meninges, etc., seem to demand some source of infection other than the trachea and bronchi.

Sometimes in cases of chronic phthisis an excess of pigment is noticed in the skin and mucous membrane (not cyanotic, but resembling Addison's disease). As such changes are known to be associated with tuberculosis of the supra-renal capsules in Addison's disease, it is reasonable to suggest that in the cases referred to tubercle has found its way into the adrenals, or some ductless glands or hæmapoetic tissue, which would thus account for the abnormal pigmentation.

A superabundance of good air is the most important element in treatment. The action is not purely local upon the tubercle in the lung, but general through the blood. We see this from the effect of open-air treatment in tubercular affections of bones, joints, etc. The aeration of the blood is the point aimed at. This may be encouraged by respiratory exercises. The

Digitized by Google

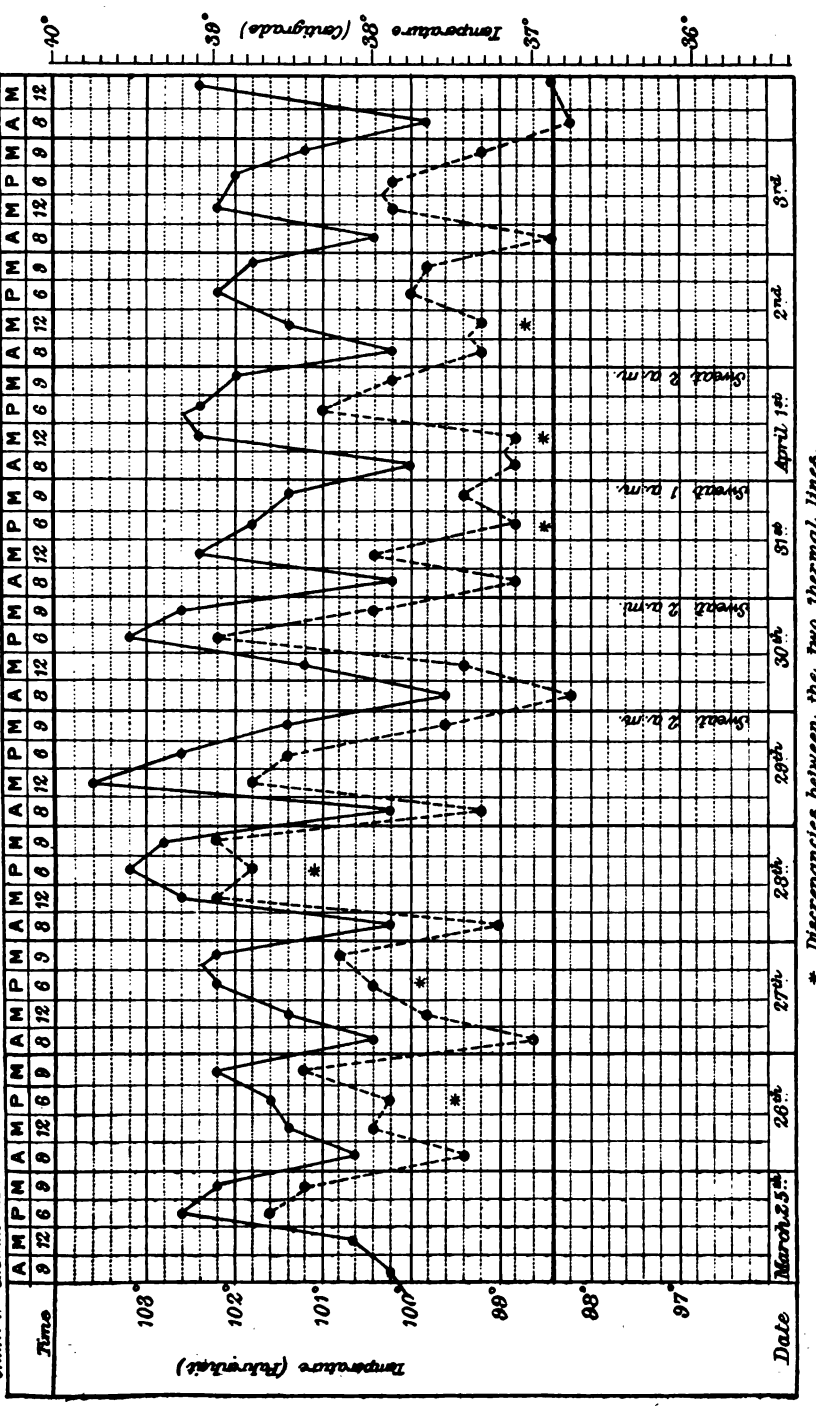
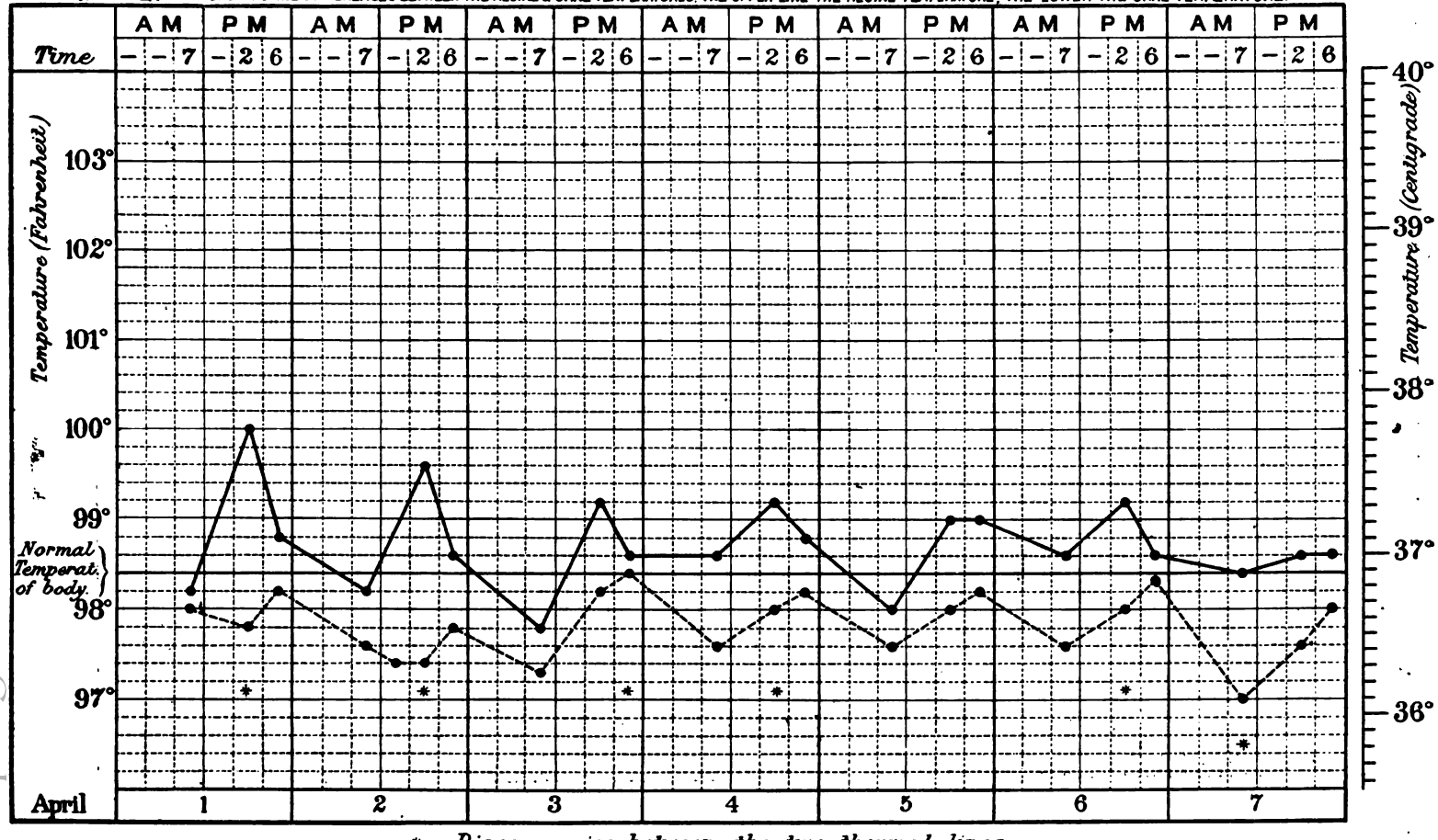


CHART I. SHOWING THE DIFFERENCES BETWEEN THE RECTAL & ORAL TEMPERATURES. THE UPPER LINE THE RECTAL TEMPERATURE, THE LOWER THE ORAL TEMPERATURE.

* Discrepancies between the two thermal lines.

Digitized by Google

CHART 2. SHOWING THE DIFFERENCES BETWEEN THE RECTAL & ORAL TEMPERATURES. THE UPPER LINE THE RECTAL TEMPERATURE, THE LOWER THE ORAL TEMPERATURE.


A M P M A M P M A M P M A M P M A M P M A M P M A M P M
Time – – 7 – 2 6 – – 7 – 2 6 – – 7 – 2 6 – – 7 – 2 6 – – 7 – 2 6 – – 7 – 2 6 – – 7 – 2 6
Temperature (Fahrenheit)
103°
102°
101°
100°
99°
Normal Temperat. of body.
98°
97°
Temperature (Centigrade)
40°
39°
38°
37°
36°
April
1
2
3
4
5
6
7

* *Discrepancies between the two thermal lines.*

Digitized by Google

fondness of German doctors for restraining movement is in many cases carried to excess. The value of the Sandow and other gymnastic exercises is great. Respiratory exercises should be encouraged, as tending to complete blood aeration, as soon as the temperature justifies their use. This must be carefully watched, and the exercises must be regulated in accordance with the indications of the thermometer.

The charts that accompany this paper, in which rectal and mouth temperatures are both recorded, show that the variations in the mouth are not sufficiently reliable to justify their use as a guide to treatment. The rectal temperatures can be depended upon, and have proved and are proving of essential value as guides to treatment.

Dr. Wills, whose experience in the management of cases of phthisis under the open-air treatment is large, has kindly, at my suggestion, put in writing his views on the value of rectal temperatures.

The accompanying charts might, at first sight, be held to prove the needlessness of taking temperatures in the rectum, but frequent serious discrepancies in the ratio between these and the temperatures taken in the mouth will be found on observing them closely; and when it is borne in mind that the diurnal variations in the temperature are almost the sole factors on which reliance can be placed for the daily guidance of the physician as to the amount of exercise or rest to be ordered, such discrepancies will no longer appear of trivial importance.

Moreover, in the subjoined charts the mouth temperatures have been taken with especial care, and compare very favourably as to accuracy with those taken in the ordinary casual way by mixed classes of patients, many of whom are persistent mouth-breathers. For ordinary clinical purposes, no doubt mouth temperatures suffice, and are justified by the ease and rapidity with which they are obtained; but then we have other and rapidly-varying factors to guide us in our direction of the patient.

It is worth noting that although much difficulty might be anticipated from the repugnance of the patient to this method, it is very rare indeed to find any objection raised by patients who are undergoing sanatorium treatment if the reasons in its favour are fairly and properly explained to them.

It has seemed worth while to submit these few remarks on this subject, because the value of rectal thermometry has lately been challenged, but sanatorium experience is so unanimous in its favour that I think the objection can hardly be sustained. I have nothing new to offer with regard to the effect of exercise, excitement, or any form of fatigue on the temperature, but it is obviously as necessary to us to have accurate information on these matters as it is to a sailor to have an accurate barometer.

Chart 1 gives the temperature as tested simultaneously in the mouth and rectum every four hours, from which it appears that when the pyrexia is considerable, the temperature as taken in the rectum differs by about a degree, and sometimes by two degrees, from that taken in the mouth.

Chart 2, on the other hand, shows that when the temperature in the mouth habitually is sub-normal, that in the rectum, though occasionally

Digitized by Google

sub-normal, frequently rises to about 99 degrees, and gives useful information to guide treatment.

After exertion, when the mouth temperature is unaffected, it is not unusual to find a marked temporary elevation of rectal temperature; this, if quite transient, is not perhaps of much moment, but affords useful indications as to the amount of exertion which may be taken with impunity.

It has been asserted that *wind* is deleterious to phthisical patients, and my friend Dr. Gordon, of Exeter, has collected an interesting series of observations in Devonshire in support of this contention. The special prevalence of phthisis in wind-swept places may perhaps be due to the increased desire to exclude wind; thus natural ventilation is prevented, and the rebreathing of polluted air brings its natural consequences. A careful observation of the influence of wind on patients subjected to the full effect in windowless rooms, on balconies and in the open air, sets aside this fear.

Wind is annoying, chilling, and, if accompanied by dust, is uncomfortable, and in all these respects is, of course, objectionable, but on the other hand it sweeps away noxious emanations and supplies fresh air as nothing else does.

Some are so impressed with the evil effects of wind that they cannot face a gentle breeze without complaining of shortness of breath, who can yet climb considerable hills with ease, the difficulty being, to a large extent, imaginary. When it is realised that such is the case, and that wind is salutary, these same patients become as pleased with themselves and as manifestly benefited by exposure to a quickly moving large body of air as they are to life in all weathers without windows. A draught—*i.e.*, air travelling rapidly through a narrow chink—is very dangerous, but exposure in an open place to a strong wind is beneficial.

A series of careful investigations as to ventilation in the rooms of the Ventnor Hospital show that with open windows the amount of carbon dioxide is much the same as in the air outside, viz., about ·035 %; with windows half open the amount was found to be ·064 %; with window-sash open at the top only, about ·073%. The experiments were all made between 7 and 7.30 A.M., before the patients were up.

It is interesting to note that notwithstanding the similarity of the air, as judged by chemical analysis, inside and outside the room, there is yet a marked difference in the effect of treatment when the patient is able to live by day and night in a shelter, a balcony, or in the open air. This difference can scarcely be due to the increased purity of the air, but rather to the fact that in the presence of wind or air in movement the gain is greater, and the improvement more marked, than when the air is stationary. The wind may not be strong, but it is sufficient to produce a greater effect than that of air in a room. It would seem that in the open air the patient does not long maintain around him his own atmosphere, that is, the air as vitiated by his own bodily exhalations.

The selection of a suitable environment must ever be a matter calling for special discrimination and wide experience. Notwithstanding the progress made in our open-air institutions, there are still a good many cases in which

Digitized by Google

foreign health stations are so manifestly indicated that we cannot withhold from our patients the benefit of such havens; there are, however, a large number of cases which a few years ago would have been subjected to foreign climate treatment, which can now be treated with at least as much success without expatriation.

This paper is chiefly based upon cases under the care of Dr. Wills, of Bexhill, and Dr. W. Gardner, of Bournemouth, who have kindly supplied some of the accompanying notes, and by whose assiduous care and watchfulness the certainly remarkable results have been obtained. (Both of these gentlemen happily are present, and prepared to give any further details that may be desired.) Of the cases thus detailed some are slight and some severe.

If the digestion is faulty, the nutrition defective, the power of exertion lessening, even although there should be no definite evidence of tubercular mischief in the lung or throat, a few weeks spent under supervision in an open-air home, with much recumbency, plenty of milk, and high, if not forced feeding, will result in the threatened evil being more quickly and certainly arrested than by a fatiguing trip to Scotland or Switzerland, or a loiterous sojourn in the Riviera.

At the other end of the scale we have cases of this type:—

A. B., aged thirty-eight, bad family history, engrossing and responsible business, home anxiety (sick wife and children), unnoticed but increasing emaciation, night sweats, loss of appetite, insidious and extending lung disease. When first seen, temperature 101°, pulse 140, power of assimilation nil, unable to take anything. At the end of a week spent in bed in an open-air nursing home, able to take three ounces of milk every two hours. In two months able to walk a mile; in six months started for South Africa to commence business, considering himself well, having gained two stones in weight.

C. D., aged eighteen, case of galloping consumption, with throat complications and high fever. Under nursing-home treatment, night sweats ceased, fever arrested in three weeks. Weight gained. Loyally carried out every detail of treatment, and is now convalescent.

A similar case treated at home gained much when the windows were taken out; the fever quickly ceased and has not since returned. This case is of interest: rapidly advancing disease in the lung, with high fever, wasting, night sweats, and profuse nummular expectoration. Under open-air methods carried on at home, in Sussex, under the eye of a devoted sister and highly capable medical man, the acute phase of the disease was speedily arrested.

E. F., aged sixty, a case of very chronic progressive phthisis, with cavity at apex. Tubercle bacilli abundant in expectoration, but associated micro-organisms few. Benefit gained during three months spent at St. Moritz. Soon after return to England cough and other symptoms increased. Disease progressing, in October, 1899, went to Bexhill Home. Under formalin spray, creosotal (in capsules), and open air, improvement set in. Physical signs showed limitation of disease; weight increased twenty pounds. The disease was checked, infiltration cleared up, and a chronic dry cavity remained.

This patient spent several months at Nordrach-on-Mendip, where steady improvement was maintained. He increased in weight, and became

Digitized by Google

two stones heavier than before his illness, and by persistence in open-air methods is now able to live comfortably in his own house.

He found the "Nursing Home" less entertaining than the "Institution" treatment, the variety of patients being greater. At first the quiet and watchful care of the home was most advantageous, but when he was able to enter into the social life of the sanatorium, he found less monotony and weariness than in the smaller home.

Miss W., *æt.* thirty-six, admitted June 10th, 1901. No tubercular trouble among immediate family. Enjoyed good health until fifteen months ago. First sign was hæmoptysis. Previously had been tired and out of health, but without cough. Cough supervened, and continued through last summer. At that time right lung was affected, but she got quite well by December. In January caught fresh cold and had hæmoptysis again, and again in March. Has lost a little weight.

Right chest flattened. Resonance not impaired. Respiration cog-wheel, some crepitant sounds at extreme apex. Voice conduction increased slightly and marked whispering pectriloquy.

Left resonance impaired below clavicle, particularly at inner end of second spaces and in axillary region.

Loud mucus râles all over upper lobe, but no increase of voice conduction.

Loud pleuritic rub over fourth and fifth spaces in anterior axillary line. Has had considerable pain there.

Pulse, 96. Height, 5 ft. 4 in. Weight, 7 st. 8 lb.

Since admission to Bexhill Home, June 28th, has gained 1½ lb. Feels greatly improved. Has been sleeping in balcony all night for three nights.

Temperature first three nights after admission, 103.5°. Dropped to 100 degrees; now slightly higher—*viz.*, 101.4°.

A further transient exacerbation of fever has recently occurred.

F. D., *æt.* twenty-six. Clerk. Good family history and good health until (when in the United States in February, 1901) he was exposed for three hours when out duck shooting. The boat was upset, and when picked up the patient was unconscious. Pneumonia followed.

First seen in 1899. Consolidation of the right lung and some softening of the left apex was noted.

Admitted to Bexhill Home, where the cough and fever ceased. He gained a stone in weight in three months; the local signs were limited. Temperature normal or sub-normal. Pulse hurried on exertion. Disease quiescent, but not permanently arrested. Pinewood, the London Open-Air Sanatorium near Wokingham, suggested, and South Africa in winter.

P. B., *æt.* twenty-nine. Good family history. Cough seven years. Under Dr. Coghill, of Ventnor, improved. Illness returned when in business at Lloyds. At a sanatorium in North Wales was subjected to the "forced feeding" system common at Nordrach. The digestion, previously good, became troublesome, and has so continued. Sea air at Bexhill caused sleeplessness. Arosa did no good, though he spent the winter there, at an altitude of 6000 feet. Is returning to Ventnor, and afterwards to Malvern, and, for the winter, Algiers and the Desert at Biskra.

Digitized by Google

Mrs. M., *æt.* thirty-eight. Phthisis following prolonged anxiety. Open-air system carried out at her own home in Bucks, under skilled supervision. Fever arrested. Tubercle bacilli ceased in expectoration, a stone in weight was gained, and she is now better than for years and convalescing at the seaside. The progress, though good, seemed somewhat slower than at a nursing home.

Mrs. W., *æt.* forty-nine. Sleeplessness following great nerve wear. Cough incessant. Respiratory sounds at the apex faulty, slight afternoon fever.

Nursing-home treatment adopted with a view of removal from home cares. Long quiescent lung symptoms tending to develop afresh, throat very sensitive with secretion from the pharyngeal membrane.

H. J. C., *æt.* twenty-six. Hæmoptysis two years ago. Pain beneath left clavicle and cough; now consolidation at the apex with softening. In the home at Bournemouth gained 15 lb. in three months. Moist sounds ceased. No tubercle bacilli ever found. Returned to his work as an auctioneer, lost ground, and has decided to give up work for two years and live at Bournemouth.

J. T., *æt.* twenty-two, a narrow-chested, badly developed man. Height, 5 ft. 8 in.; weight, 9 stone. Chest circumference, 31–33 in.

At left apex respiration wavy; at right supra spinous fossa bronchial breathing and bronchophony. Vocal cords congested. Some gain at Bournemouth; on return to Eastbourne no tubercle bacilli to be found in expectoration.

Mrs. Z., *æt.* twenty-nine. Weight 10 st. 7 lb. on admission; 11 st. 12 lb. after two months' treatment in Bournemouth Home. Hæmoptysis in October, 1898. Tubercle bacilli excessive, disease limited to the apex and inter-arytenoid space. This last was treated with lactic acid, and subsequently scraped by Dr. G. Macdonald. Tubercle bacilli diminished, general condition improved.

R. F. G., *æt.* thirty. Severe hæmoptysis, October, 1891, recurring in 1893, 1895, and 1896; purulent expectoration for three years. In October, 1898, weight, 9 st. 3 lb.; in March, 1899, 12 st. 9 lb., a gain of 3 st. 6 lb. in five months. The softening at the apex was no longer present, but dry cavernous sounds were noticed. Temperature sub-normal, tubercle bacilli present but not numerous in February, 1899, no tubercle bacilli to be detected, cavernous sounds limited, no moist sounds; chest circumference increased from 35–36 to 36–38 inches.

A. T., *æt.* thirty-four. Ex-police constable. Married. Family history good. Present weight, 11 st. 5½ lb.; height, 5 ft. 11 in. Influenza, pneumonia, pleurisy, phthisis rapid. Under open-air treatment in balcony of St. Thomas's Hospital, with high feeding, put on 13 lb. in 15 weeks. Then at Police Sanatorium at Harrogate improved; now (July 1st, 1900) going to continue open-air treatment at Ventnor Hospital. Disease arrested. Pulse 72, no fever. Phthisical signs slight.

T. A., *æt.* twenty. Cough and hæmoptysis in August, 1900, also in September, with nummular expectoration. Height, 5 ft. 11 in.; weight, 11 stone. Farmer; lived in damp house. Father phthisical, also brother. Vocal cords

Digitized by Google

inflamed; lung sounds negative. Temperature, 101° to 102° every night, sub-normal in the morning. Gained a stone in weight during first month, but signs of consolidation at right apex. Weight, 12 st. 1¼ lb.—a gain of 15 lb. in five months. The bacteriological report was very interesting:—

October, 1890. Very numerous.
December, 1890. Fairly plentiful.
January, 1901. Few in number.
March, 1901. Few in number.
April, 1901. Could find no tubercle bacilli.

P. M., *æt.* twenty-three. Height, 5 ft. 8 in. Consolidation of right lung, with small vomica at apex; slight hæmoptysis. Admitted to Bexhill Home October 7th, 1899. Under formalin, creosotive, and open-air methods, fever and pulmonary symptoms reduced; much depressed by family troubles; left home slightly benefited after three months. Then, under inhalation treatment, arrest occurred, and disease is now (July, 1901) quiescent.

J. P., *æt.* twenty-two. Mother and mother's brother phthisical. Twelve years ago had blood-spitting and congestion of the lungs; two years ago pleurisy; progressive weakness in spring of 1900; dry throat; violent cough. On admission to Holme Chase, Bexhill, height 6 ft., weight 9 st. 8 lb. Consolidation and softening of the right and, to a less extent, of the left lung. The physical signs became less marked; but some fever with symptoms of (? tubercular) testiæ and pustulæ. Two or three recurrences of severe pyrexia, with evidence of further softening in the lung, occurred, notwithstanding the rigid continuance of the open-air methods. The windows were removed, and throughout the winter, though the winds were boisterous and rain often beat into the room, the windows were never replaced. In the spring the appearance of the patient was greatly improved; he was able to drive himself about. The temperature was perfectly steady, normal or sub-normal. No marked gain in weight; disinclination for milk always a marked symptom. Patient now with his friends in the country; physical signs much more satisfactory, but strength still deficient.

Major G., *æt.* thirty-four. Sent to hotel at Bexhill in October, 1900, being unwilling to enter the nursing home. Cough six months; tubercle bacilli numerous; considerable hæmoptysis occurring, the patient was removed to Holme Chase. Evidence of consolidation with moist sounds at the right upper lobe and extending to base. In four months cough and expectoration ceased; weight gained, 1 st. 8 lb.; only physical sign present slight dulness at the right apex, thought to be due to old threatening when young. Patient looks florid, the picture of health, and considers himself stronger than ever. He now writes that he is quite well and full of activity.

The CHAIRMAN (Sir J. W. MOORE): The Section has listened with great interest and pleasure to this admirable paper. I was delighted personally to hear Dr. Symes Thompson speak as to the innocuousness and harmlessness of wind. I think the danger of wind is very much overestimated, and, as regards that, we can escape from winds; and as soon as we make up our minds to

Digitized by Google

endure it, the better. Dr. Squire is anxious to address the Section in connection with this matter.

J. EDWARD SQUIRE, M.D. (*London*): I have had now a good many years' experience of what I always preferred to call the "hygienic" treatment of consumption, although I am quite ready to adopt the name "sanatorium" treatment. In the hospital to which I am attached (the North London Consumption Hospital) we have for many years preached and practised the hygienic treatment of consumptives, and as soon as public opinion was educated up to realising that there was no danger in night air we were able to carry out the "open-air" treatment. In other words, the patients, instead of being out only during the day, were out during the night as well. One point that must strike everybody is the necessity for discipline, if you are going to do any good with patients in this hygienic treatment. By that I mean that we have to teach the patient how to regulate his life to the best advantage, and then to ensure that he carries out the instructions which are given him. This generally requires constant supervision, and is easier in a sanatorium, where regulations are laid down and rigidly enforced, than in a nursing home, where the patient expects more freedom. That is the one point where, in these nursing homes that Dr. Symes Thompson speaks of, we might possibly have some difficulty. It is not all men, and perhaps still fewer women, who have the wish—even if they have the power—to coerce patients against their will. We know what a nurse can do with refractory patients, but when it comes to a person who is not too ill to be a sort of companion rather than a patient, it is most difficult, week after week, to coerce that person into doing things which are sometimes distasteful, but which must be carried out if we want to get the full effects of the treatment. In different sanatoria, though in all there are strict regulations, the details of the regulations differ even in what seemed essential points. In one forced feeding is insisted upon, in another this is not required, but graduated exercise is everything; and in another neither of these are the points upon which you are to rely, and something else is put down as essential. It must have struck most of us that when so much stress is laid upon one detail of treatment at one place, and an equal stress on some quite different detail at another place, there cannot be much use in these regulations, because at the one place patients do as well as at the other. But when we come to look into the matter we find that the underlying principle is sound. The important point is that there are rules which have to be obeyed, and, apart from the exact details contained in the rules, the obedience to regulations is in itself a valuable training. In order to get people to do what they are told you must lay down certain rules from which you will allow no departure whatever. It matters little, to my mind, what the rules are, so long as they are not injurious, because the result of insisting upon the following out of certain rules gives a disciplinary education which is, after all, of essential importance. Whether you oblige a man to obey rules about feeding or about walking, the effect is in either case to teach him to do what he is told, and when he has once learnt that then you can

Digitized by Google

send him to a nursing home or to his own home to carry on the treatment. I think we all know the tale of the old soldier who got so into the habit of doing exactly what he was told that when, years after he left the service, he was going home one day with a parcel, somebody in the street called out "Attention!" to him, he came to attention and dropped the parcel in the road. He had no time to think, but had got into the habit of obeying an order, and he acted on that habit. Having instilled into the patient the habits of discipline—the ready obedience to authoritative orders—it was possible to relax the strict supervision of the sanatorium, and the treatment could then be satisfactorily carried out in a nursing home or in the patient's own home. There is a further point to that which I think worth referring to again. It was mentioned yesterday that the keeping up of the temperature of a room when you have got all the windows open is a matter of difficulty. And, if it is of importance, how are we to get over it? Now in the North London Consumption Hospital the difficulty was found in the colder weather in keeping the ward temperature up to anything like what the rules of the hospital said they ought to be kept up to, and, in fact, the difficulty was insuperable. It was impossible by the heating apparatus that there was at the hospital to keep up the temperature of the wards to anything like what was supposed to be necessary, so long as the windows were wide open. One day I went up to the hospital in the colder weather, and I said: "Is it not cold enough to have a fire?" and they said, "Oh! we have given up fires in the wards. It is no good, and the patients say they do not mind whether it is cold or not, so we have given up fires and leave the wards as they are." As a matter of fact, we have found that it does not matter about the wards being much below the temperature that we consider comfortable, for the patients are warm in bed, and, although the nurses do not like it, the patients who are in bed keep themselves comfortably warm and do quite as well with the temperature low. So that we need not trouble, I am sure, about attempting to keep up the temperature so long as the patient himself is kept warm in bed. The latter I do think is important. It has been said by one speaker that, in his experience, children do not do well with sanatorium treatment. Now, I think a very great deal depends upon the exact age of the young patient. Children of ten or twelve years of age do, in my experience, remarkably well and often get over an amount of lung mischief which an adult would probably not be able to recover from. When the age is a little above that—when, in fact, puberty commences—then what reserve energy there was in the young individual is expended in another direction, and cannot be utilised to get rid of the tuberculosis. I have for long noticed that children who have nearly but not quite reached puberty frequently recover from a degree of tuberculosis of the lungs which would give no hope in an adult. It has seemed to me that a reserve of energy, stored up to bring about the developmental processes of that period, is utilised in resisting and overcoming the tuberculous attack. In these cases puberty is delayed. If, however, the developmental changes have already progressed towards completion, the patients do very badly—they have not enough energy to complete puberty and to withstand tuberculosis

Digitized by Google

at the same time. A girl of, say, twelve or thirteen, who is still in all respects a child, has a good chance of recovering from active tuberculosis of the lung, whereas a girl of about the same age who, even though menstruation may not have commenced, shows evidence of reaching the stage of puberty, would, with an equal amount of mischief in the lung, have much less expectation of improvement.

M. LE DR. LASALLE, *de Bordeaux, délégué de la Ligue Française contre la tuberculose.*

JE n'avais pas l'intention de prendre la parole dans cette section. M. le Dr. Armaingaud, le fondateur des sanatoriums marins d'Arcachon et de Banyuls-sur-mer, le fondateur et le président de la première Ligue contre la tuberculose, devait vous présenter lui-même quelques observations; mais, empêché au dernier moment d'assister à ce congrès, il vient de m'écrire de vouloir bien le supplier, et de vous soumettre, en son lieu et place, quelques réflexions.

Dans la prophylaxie de la tuberculose on paraît oublier un peu en ce moment le rôle éminemment utile des sanatoriums marins; on paraît méconnaître qu'ils sont pour l'enfance débile, surtout pour les enfants issus de parents phtisiques, la meilleure, la plus sûre sauvegarde contre l'envahissement et le développement bacillaire. Depuis quelque temps l'attention du corps médical est presque exclusivement absorbée par les sanatoriums tuberculeux. Loin de moi la pensée de diminuer leur rôle bienfaisant, nécessaire, mais j'estime que les sanatoriums tuberculeux ne doivent pas faire négliger les sanatoriums marins.

Les premiers constituent un traitement curatif; les seconds un traitement préventif; or c'est ici le lieu de rappeler la vieille maxime: mieux vaut prévenir que guérir. Voilà la première observation que mon éminent ami Armaingaud voulait vous présenter. Permettez-moi en quelques mots très brefs de vous en soumettre une seconde.

Messieurs, le but des organisateurs de ce beau congrès n'a pas été seulement de provoquer des discussions scientifiques, mais encore, j'allais dire surtout, d'étudier, d'adopter, et de préconiser toutes les mesures d'ordre social qui nous paraîtraient utiles pour lutter contre le redoutable fléau. Il faut bien le reconnaître, messieurs, si les découvertes de Villemin et de Koch nous ont merveilleusement éclairés sur la nature de le phtisie, elles n'ont pas fait encore surgir un traitement spécifique; non, messieurs, nous le savons tous, il n'y a pas une thérapeutique spéciale à la tuberculose; et s'il est vrai que sur quelques points particuliers nous ayons trouvé quelques médications utiles (mon très distingué compatriote M. Hobbs nous en a fourni la preuve il y a un instant), nous devons reconnaître que c'est l'hygiène, surtout l'hygiène, qui nous a donné jusqu'ici des résultats certains. De cette proposition indiscutable découle cette nécessité absolue, qu'il faut éclairer le public, l'éclairer complètement, si nous voulons lutter victorieusement contre les ravages de la tuberculose. C'est ce qu'a compris l'un des premiers

Digitized by Google

le Dr. Armaingaud, qui a fondé sa ligue contre le phtisie pulmonaire, ligue qui a trouvé des imitateurs dans presque toutes les nations civilisées.

Travaillons, donc, messieurs, à faire l'éducation du peuple, enseignons lui l'hygiène preventive et curative de la tuberculose. C'est l'œuvre la plus pressante et la plus féconde que nous puissions accomplir. Comme l'a dit quelque part le professeur Landouzy, avec son éloquence entraînante: "Il est temps d'en finir avec cet opportunisme médical qui, pour ne pas effrayer les gens, les laisse sans défense puisqu'ils sont sans défiance contre l'une des plus terribles maladies qui désolent l'humanité." Ayons le courage de détourner le peuple de tous les prétendus spécifiques qu'une réclame offre sans cesse à son ignorance et à sa crédulité, au grand détriment de son estomac et de sa bourse. N'oublions pas enfin que sans le concours de l'opinion publique il nous serait presque impossible de réaliser les réformes que nous réclamons. Ce n'est que par cette association d'efforts entre le public et nous que nous pouvons enrayer et faire disparaître cette peste cruelle qui, comme le disait hier le professeur Brouardel, compromet le plus gravement l'avenir de nos patries.

SANATORIUMS ET VILLAS SANATORIA.

PAR M. LE DR. BARADAT, *de Cannes.*

UN Congrès, tenu en Angleterre, dans le pays qui envoie le plus de personnes aux contrées d'air pur et de soleil, était naturellement amené à donner une place importante, dans ses travaux, à la question des Sanatoriums. J'ai tenu à vous parler du système de la "Villa Sanatoria." Ce sujet m'intéresse personnellement, puisque, dans les différents congrès qui ont précédés celui-ci, notamment au congrès de Berlin de 1899 et au XIII^e^ congrès international de Paris de 1900, j'ai soutenu la cause que je viens développer aujourd'hui. D'ailleurs, j'ai eu la satisfaction de rencontrer parmi les défenseurs de la cure libre des maîtres d'une autorité universellement reconnue. Je n'aurai le plus souvent qu'à citer leur opinion.

Vous savez, Messieurs, que deux systèmes de Sanatoriums sont en présence. Le *Sanatorium fermé* où les malades, retirés à leur famille, se trouvent réunis ensemble dans une sorte d'internat, soumis à une même règle sous la direction d'un même médecin, et le *Sanatorium libre* appelé "*Home Sanatorium*" par le Professeur Landouzy et qu'on pourrait aussi bien nommer la "Villa Sanatoria." Le malade y vit à part des autres malades, dans l'isolement de son château ou de sa villa, entouré de sa famille.

Je vous rappellerai brièvement, Messieurs, les inconvénients du Sanatorium fermé. La cuisine faite pour un grand nombre d'individus est forcément plus négligée, les mets moins bien préparés, moins adaptés aux besoins de chacun, à des estomacs souvent fatigués et bouleversés. Or, la question de nourriture a une importance capitale dans le traitement d'une maladie où l'alimentation et la suralimentation jouent un si grand rôle. De plus, dans le Sanatorium fermé, le malade a un grand ennemi, l'ennui. Il commet les pires imprudences pour se soustraire à la monotonie de la vie agglomérée. S'il a l'habitude

Digitized by Google

de boire, il continue à chercher dans l'alcool une excitation dangereuse. Il va et vient au gré de son caprice et se trouve de la sorte à la merci d'un refroidissement. La promiscuité du Sanatorium lui donne mainte occasion de flirt qui souvent l'entraîne à des excès débilitants, bien qu'il aie un impérieux besoin d'économiser ses forces. Une forte discipline serait la seule garantie contre de pareils dangers, mais les pancartes affichées sur les murs et qui étalent, dans une belle ordonnance, la suite des règles à observer à chaque heure du jour sont pour la plupart des malades de simples mots sans force. L'autorité du médecin serait indispensable au Sanatorium, mais elle en est le plus souvent absente. Ces établissements sont trop souvent livrés à des Directeurs commerciaux qui laissent fléchir la discipline devant la perspective de gains rapides et de gros dividendes. Ils donnent à l'alcool, sous toutes ses formes, un large permis de circulation ; ils ferment les yeux sur le flirt et ses suites ; ils laissent aller et venir leurs malades ; ils sont loin de se condamner eux-mêmes à une fatigante présence de tous les instants, et le Sanatorium, conduit d'une main indolente, entoure le malade de périls au lieu de le rendre à la vie.

Messieurs, je m'oppose au Sanatorium fermé au nom des mêmes principes qui m'ont fait rejeter l'internat en matière d'éducation. Je hais l'agglomération, je hais l'encombrement dans un même espace, d'individus même sains, à plus forte raison de malades, qui, par leur ensemble, créent un milieu bacillaire pernicieux. Dans la question du Sanatorium, comme dans celle de l'éducation, je suis contre le système allemand et suisse qui lutte contre la maladie par l'encasernement des malades, et pour le système anglais qui lutte contre elle, comme dit le Professeur Grancher, par le bifteck et le tennis.

Pour toutes ces raisons, il ne devrait y avoir que des Sanatoriums fermés pour impulsifs, pour des malades incapables de pouvoir se diriger ou d'avoir une volonté, et des Sanatoriums fermés pour pauvres. Dans ces derniers surtout, la discipline hygiénique y sera toujours rigoureusement maintenue parce qu'elle ne trouvera pas d'obstacle dans la complaisance intéressée d'un Directeur. Les hommes y seront séparés d'avec les femmes et le pauvre y trouvera une alimentation mieux préparée que chez lui, un milieu quelquefois plus aéré, et le repos. Ces asiles en pleine campagne lui donneront l'espérance toujours, et la guérison assez souvent.

Les difficultés du Sanatorium commun ont fait naître l'idée du Sanatorium personnel auquel se sont ralliés les premiers les Professeurs Landouzy et Lemoine de Lille, le Dr. Lalesque, d'Arcachon, et le Professeur Brunon, par son système de la cure libre en Normandie. Les principes essentiels y sont appliqués mais les inconvénients du Sanatorium fermé disparaissent. Sans étaler l'attirail de chauffage et d'aération de ces internats de guérison, de ces casernes de tuberculeux, la maison de campagne, cabane ou château, laisse entrer l'air dans les chambres par les fenêtres largement ouvertes, les chauffe le jour au soleil et la nuit à un clair feu de bois. Chez lui, dans son home, le malade s'alimente, à son goût, avec une nourriture saine, bien préparée et dont il a l'habitude. La vie de famille le préserve de l'ennui, il n'a pas besoin d'alcool ni d'allées et venues dangereuses, ni de distractions énervantes. Il se trouve dans les meilleures conditions possibles pour observer l'hygiène et

Digitized by Google

pour goûter le repos. D'ailleurs il ne faudrait pas croire que dans le Sanatorium libre l'absence d'un Directeur surveillant se fasse sentir au préjudice du malade. Ici le médecin remplace le Directeur, il est le plus souvent l'ami de la maison, il remplace par son influence morale et toute proche l'autorité disciplinaire qu'exerce, de haut, le Directeur sur une collectivité. Il peut, comme le dit M. Lalesque, régler l'alimentation du malade, le soumettre au repos, cuber l'air qui convient à ses poumons et ordonner les exercices appropriés à son degré de force ou de résistance. En outre, plus accoutumé que le Directeur du Sanatorium au tempérament individuel de chacun de ses malades, il connaît mieux le genre de thérapeutique qui lui convient, et vous savez, Messieurs, que le Sanatorium, selon la parole du Professeur Landouzy, "n'est pas par elle seule la médication de la tuberculose pulmonaire comme la quinine est le spécifique de la malaria." Les médicaments appropriés à chaque malade sont choisis et appliqués par le médecin.

Il resterait à nous demander, Messieurs, dans quel séjour il faudra de préférence placer la Villa Sanatoria. Il y faut une réunion de circonstances utiles, un air pur, sec, peu excitant et un ensemble heureux de circonstances atmosphériques qui permettent au malade de jouir de cet air le plus possible. Il y faut la lumière et le soleil, ces grands destructeurs de bacilles dont l'action bactéricide purifie l'air et le sol.

Une région en France présente assemblées toutes ces qualités salutaires, et c'est ici, Messieurs, que j'aurais scrupule à parler en mon propre nom. Je cède volontiers la parole, pour vous dire l'action bienfaisante de l'air méditerranéen au Professeur Landouzy: "C'est sur la Rivière," dit-il, "mieux qu'ailleurs que le tuberculeux pulmonaire, le prédisposé à la tuberculose, peuvent établir leur Home-Sanatorium, en un lieu où la température est de faible écart entre le jour et la nuit, et où des installations bien construites, hygiéniquement comprises, aseptiquement gouvernées, bien orientées, sont garanties aux malades. Il faut moins de luxe que de confort sanitaire pour réaliser la cure hygiéno-diététique. Là, sur la Rivière, le tuberculeux trouve dans l'ensoleillement de sa résidence aussi bien que dans l'air de la mer qu'il respire et dans l'aspect riant de la campagne qui l'entoure, de quoi se réconforter et tromper son ennui, sans excitation factice. Willis, au XVII^e^ siècle, envoyait déjà dans ce coin de la France ses compatriotes pour lesquels il redoutait les épais brouillards de la Tamise. C'est là que James Henry Bennet, brisé, au fort de l'âge, par la tuberculose pulmonaire, abandonnant Londres, vint se réfugier. C'est de là qu'il a appris aux médecins des deux mondes toutes les ressources que le climat méditerranéen met au service des tuberculeux."

J'ajouterai, Messieurs, que l'illustre Chancelier d'Angleterre, Lord Brougham, fut le Christophe Colomb de Cannes, qui est demeurée la résidence la plus aristocratique de la Côte et le quartier favori des Anglais.

Tous ceux qu'attireront l'excellente salubrité de notre littoral méditerranéen, la vision charmante de la grande Mer Bleue, tous ceux qui viendront chercher sur nos rives le repos, la joie et la vie se persuaderont que la Ville de Cannes eut raison autrefois d'adopter cette fière et jolie devise: "Qui li vèn li vieù" (Qui y vient y vit).

Digitized by Google

DISCUSSION ON THE USE OF THE RÖNTGEN RAYS IN THE DIAGNOSIS OF PULMONARY TUBERCULOSIS.

OPENING ADDRESS.

By HUGH WALSHAM, M.A., M.D. Cantab, F.R.C.P. Lond.,

Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, E.; and Assistant Medical Officer in the Electrical Department of St. Bartholomew's Hospital.

SINCE Professor Röntgen made known his brilliant discovery, now some five years ago, I have been at work on the use of the X rays in the diagnosis of pulmonary tuberculosis. I propose bringing before you this morning very shortly some of the results attained. The questions I proposed to answer, if possible, were the following, viz. :—

1. Can the Röntgen rays show tubercle in the lung?
2. If so, at what stage of their development?
3. Can the rays detect tubercle in the lung before the other means of physical diagnosis already at our disposal?.

Now, obviously the first step to be taken in the attempt to answer the first question was to obtain a skiagram of a normal chest, to study its shadows, and endeavour to differentiate between the shadows cast by structures outside the thorax from those caused by its contained organs. The next was to take a skiagram of an undoubted case of pulmonary tuberculosis and compare the one with the other.

I will now, first of all, throw on the screen a picture of a normal chest (see fig. 1). We see at once that the pulmonary image in health is quite transparent from apex to base, with the exception of a few ill-defined shadowy lines to the right of the heart shadow. These cardiac lines are seen more or less pronounced by all skiagrams of the healthy chest, and must, therefore, be considered normal. Now, on what do they depend? Are they caused by the division of the lower bronchi? I think not. If we look at a skiagram of a normal lung outside the chest, we see that only the primary divisions of the bronchi are seen, and, in addition to this, these cardiac lines are too low in position to be caused by the bronchi. I have endeavoured to clear up this point in the following way :—I took a skiagram of a dead body on the post-mortem table; I then removed the sternum, opened the pericardium, removed the heart, replaced sternum and integuments, and took another radiograph. The lines still remained. I then reopened the chest, removed the lungs, replaced the sternum and integuments, and again radiographed the body. The

Digitized by Google

final stage I show you on the screen (see fig. 2). You see that these cardiac lines still remain, and are caused, no doubt, by the junction of the pericardium with the pleuræ. They are much more distinct in the dead body, of course, as they are not in constant movement by the systole and diastole of the heart as they are in a living person.

The next point you will observe is the shadow of the scapulæ is absent

FIG. 1.—NORMAL CHEST.

FIG. 2.

(see fig. 1). Their shadow only complicates matters, and is better removed. This can be done in the following way: The patient lies in the prone position on the plate, the arms being extended and hanging over the end of the couch. In this position the scapulæ are turned edgewise, so to speak, and their shadow removed from the skiagram.

The next shadow I should like to call to your attention is one shown well

Digitized by Google

in this skiagram (see fig. 1), and usually seen in the more muscular subjects. It is, as you see, somewhat triangular in shape, and extends upwards and outwards towards the axillæ. By the stereoscopic method these shadows are seen to be outside the chest, and are caused, no doubt, by the anterior axillary folds. They are only seen, I think, when the patient is skiagraphed in the prone position with extended arms, this position naturally rendering the axillary folds thicker. Of course, the shadow of the pericardium with contained heart in the anterior position is well seen. I need only just mention it. The shadow of the diaphragm is also well seen. And here, in passing, I will make a short digression. It is not so much the shadow of the diaphragm that is important in pulmonary tuberculosis as its movements with respiration as seen with the screen. Now, if the diaphragm be watched with the screen, we see that it does not, as physiologists tell us, become flatter with inspiration. Its

FIG. 3.

curve is always maintained unaltered; it plunges up and down piston-wise. And now I come to a point of great importance in the diagnosis of pulmonary tubercle. The movement of the diaphragm on the affected side is much less than on the non-affected or less affected side, and this when the disease is limited to one apex. Why this should be so is hard to explain, but the fact remains.

I will now throw on the screen a picture of a case of pneumothorax (see fig. 3), showing the depression of the diaphragm by the increased pressure in the pleural cavity. If you will look again for a minute at the picture now on the screen (see fig. 1), you will see that the healthy chest in the living person is as clear as that of the dead body with thoracic viscera removed (see fig. 2), showing that any abnormal shadow seen in the skiagram is due to some

Digitized by Google

pulmonary change—in other words, the muscles and integuments of the thorax are transparent to the rays. Not so, however, with the dead lung outside the body, which casts a very distinct shadow, although transparent in the living condition. At present I cannot offer any explanation of this curious fact.

There is one other shadow I will mention, and that is, we occasionally see an ill-defined curved shadow in women with large mammary glands. Here again the stereoscopic method shows the shadow to be outside the thorax, and therefore cannot be confounded with shadows produced by pathological changes in the lungs.

I will now throw on the screen a well-developed case of pulmonary tuberculosis (see fig. 4). You see at once there is a marked difference. The clear pulmonary image is obscured by flocculent shadow, punctate in parts.

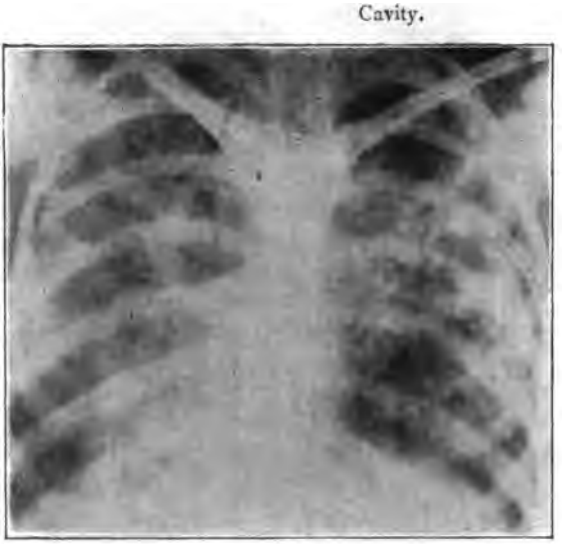


FIG. 4.

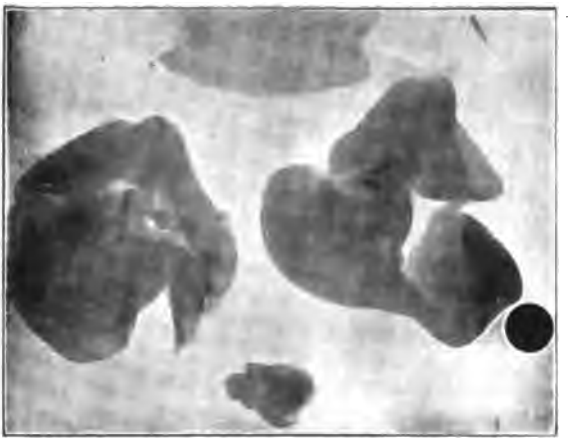
Now, on what do these shadows depend? Are they due to tubercular consolidation, or to patches of congestion or caseation or pleural adhesions, or what? I have endeavoured to answer these questions in the following way. On the screen before you (see fig. 5) is the skiagram of a tuberculous lung; nearly the whole of the upper lobe was caseous and breaking down. So we see that caseation throws a very dense shadow. The shadow lower down, caused by grey and yellow tubercle, is nothing like so dense; still, as it might be said that some of this shadow is due to blood congestion round the tubercular consolidation, I performed the following experiment. The lungs from a case of pulmonary tuberculosis were taken; one was soaked in water for some hours to break up the blood corpuscles, the vessels washed out until the water came away colourless. Both lungs were then skiagraphed, and I think you will agree with me that there is little or no difference between them

Digitized by Google

(see fig. 6). Again, it has been asserted that the blood next to the bones is the most opaque tissue of the body. With this assertion I cannot agree. Look, for instance, at the picture now on the screen (see fig. 7). It represents a portion of a rib, a piece of muscle the same size, and a blood clot. My

Caseous Shadow.

FIG. 5.



Washed-out Lung.

FIG. 6.

ye can detect no difference between the muscle and the blood clot; that is to say, clotted blood—which, by the bye, is more opaque than fluid blood—is no more opaque to the rays than muscle. Again I took the lung and

Digitized by Google

kidney which contained tubercle from the same case, and radiographed them together (see fig. 8). The tubercle in the kidney is distinctly seen, down to the minutest miliary granulation which could be detected by the naked eye. Again, look at these lungs from a case of acute miliary tuberculosis.

Bone (rib). Muscle. Blood Clot.

FIG. 7.

FIG. 8.

They show the tubercle scattered through their substance clearly enough (see fig. 9).

Putting the above facts together, we may, I think, answer our first question by saying that the Röntgen rays can show definitely tubercle in the lung.

I will now throw rapidly on the screen some cases of undoubted pulmonary

Digitized by Google

tuberculosis more or less advanced (see fig. 10). I now come to the question of the detection of cavities in the lung. That the X rays can detect with certainty a cavity in the lung is now a fact beyond question. With reference to the size of a cavity, I would say that the rays can diagnose a cavity much

FIG. 9.—MILIARY TUBERCULOSIS.

FIG. 10.—ADVANCED TUBERCULOSIS IN BOTH LUNGS.

smaller than can be detected by auscultation. The smallest I have yet met with is now on the screen before you (see fig. 11). It is less than a small marble. Occasionally auscultation gives evidence of a cavity of large size, and we are surprised, on examining with X rays, to find that the cavity is a small

Digitized by Google

one. This must mean that the consolidation round a cavity conducts the sounds produced in the cavity far beyond its limits. Furthermore, it not infrequently happens that auscultation diagnoses a cavity which the rays show has no existence.

I will now pass on to the second question, *viz.*, at what stage of development can the X rays detect tubercle? And here I am afraid we must admit that the very earliest stage in the development of the tuberculous process cannot be detected by the X rays, but this also applies to all other methods of physical diagnosis. The first beginnings of tubercle in the lung cannot be detected by this or by any other means at our disposal. There can be no doubt that the X rays are able to pick out in shadow a very small tuberculous focus in the lung. I will give a few examples. The skiagram on

Cavity.

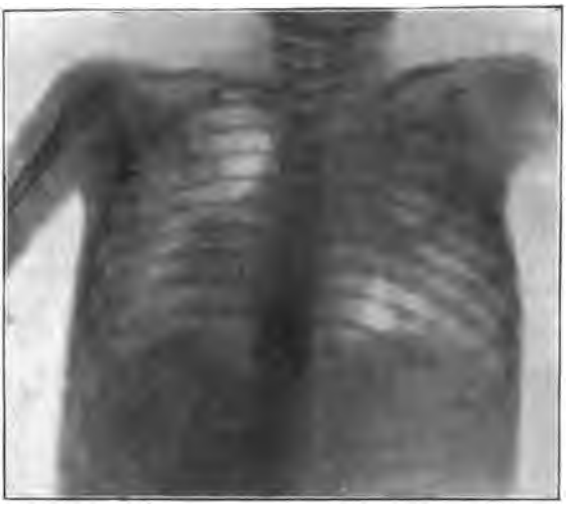


FIG. 11.

the screen is from the chest of a man aged 28 (see fig. 12); he came to the out-patient room complaining of his digestion. On examining the chest there was nothing to be made out except perhaps a little weakening of the breath sounds at the right apex. I examined this patient radiographically, and had no difficulty, as you see, in diagnosing a tuberculous process at the right apex. The next case is that of a woman aged 34 (see fig. 13). In this instance there was a little prolongation of expiration, with some slight difference in the percussion note at the right apex. I was somewhat surprised, therefore, to find that there was extensive disease in the right upper lobe. The shrinking of this side of the chest is well shown, which was not detected on physical examination. The next two pictures are both from patients in whom pulmonary tubercle was suspected, but which gave no evidence of its presence on stethoscopic examination. The shadows, however, in each case are

Digitized by Google

distinct, and later in their history the physical signs with the stethoscope became undoubted. I think, therefore, we are justified in saying that the X rays are able to diagnose really early tuberculous change in the lung. I think, also, that the Röntgen rays can detect tubercle certainly as early as the

Tuberculous Shadow.

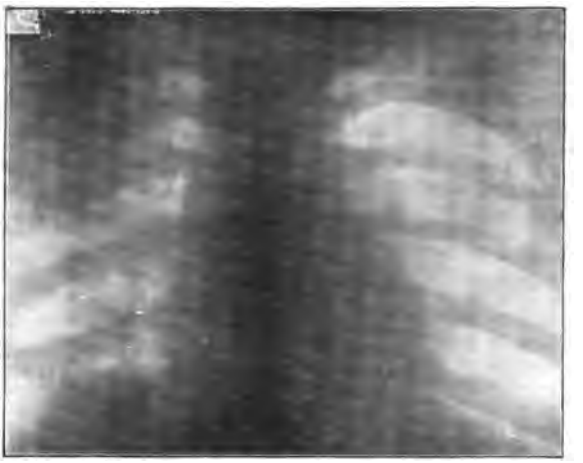


FIG. 12.

Tuberculous Shadow.

FIG. 13.

stethoscope, or even earlier, as the two last cases show. Now, it may be asked, how can you tell a tuberculous shadow from that of a new growth? This can be done only by the distribution of the shadow and by the history of the case. As with the stethoscope we have to consider the history of the individual case before us, so with the X rays. Pray do not think for one minute that the

Digitized by Google

X rays can diagnose tubercle off-hand; such, I am sure, is not the case. But I think that this means of detecting an early tuberculous shadow brings a little nearer the day when internal tubercle may perhaps be treated with chemical rays, as external tubercle—viz., lupus—is being treated to-day with such conspicuous success. In the future we may perhaps be able to say of pulmonary tubercle: "It comes as a shadow, so departs."

I will only detain you a few minutes longer; I have advisedly said nothing about methods of examination; I thought in an audience such as this it would be superfluous. I may say, however, that all the skiagrams I have had the honour to show you were taken with a coil giving a 14-inch spark between the terminals of the secondary coil. One word more and I have finished. I think in all cases of suspected pulmonary tubercle a skiagram should be taken. From an experience of over five years I am sure that a shadow can be detected on the photographic plate that would be quite missed on the screen.

SUR LA TECHNIQUE DE L'APPLICATION DES RAYONS DE RÖNTGEN AU DIAGNOSTIC DE LA TUBERCULOSE.

PAR M. LE DR. A. BÉCLÈRE,

Médecin de l'hôpital St Antoine, de Paris.

ON ne peut plus mettre en doute les services rendus au diagnostic de la tuberculose pulmonaire par l'emploi des rayons de Röntgen, mais, pour tirer de cette nouvelle méthode d'exploration et particulièrement de l'examen radioscopique le meilleur parti possible, il est bon de réaliser certaines conditions dont l'expérience m'a appris l'utilité.

Ces conditions concernent le sujet examiné, l'observateur et le matériel employé à l'exploration.

Le sujet examiné, pour éviter la fatigue et conserver la liberté des mouvements respiratoires, particulièrement la liberté des mouvements du diaphragme, doit être couché sur le dos ou, de préférence, assis sur un siège élevé, de hauteur réglable, comme les tabourets de piano, et ayant la forme d'une selle de bicyclette, de manière à permettre l'extension presque complète des cuisses sur le bassin.

L'observateur, pour être en possession d'une très grande sensibilité rétinienne à la lumière, doit demeurer pendant quelques minutes dans l'obscurité avant de commencer l'examen radioscopique. Des mensurations précises m'ont appris qu'après dix minutes de séjour dans l'obscurité la sensibilité de la rétine à la lumière de l'écran fluorescent est devenue 50 à 100 fois plus grande qu'elle n'était primitivement; elle devient environ 200 fois plus grande après vingt minutes d'obscuration et, passé ce temps, elle grandit encore.

Il est préférable que le générateur d'énergie électrique, chargé d'alimenter l'ampoule de Crookes, ait une certaine puissance: cependant il n'est pas

Digitized by Google

nécessaire que cette puissance soit très grande. En effet peu importe une différence du simple au double dans la longueur de l'étincelle que peut fournir une machine statique ou une bobine d'induction, quand c'est au centuple et deux fois au centuple que le séjour dans l'obscurité fait grandir la sensibilité lumineuse de l'observateur. J'ai décrit et j'emploie journellement une petite machine statique de Wimshurst, à quatre plateaux, simplement mise en mouvement à la main, avec une manivelle, et assez peu volumineuse pour être très facilement transportable : elle suffit pour donner, sur l'écran fluorescent, à des yeux convenablement préparés par quelques minutes de séjour dans l'obscurité, une excellente image des organes thoraciques.

En revanche il est indispensable que le pouvoir de pénétration des rayons de Röntgen, variable avec la résistance électrique de l'ampoule de Crookes, soit, toutes choses égales, exactement adapté à la sensibilité lumineuse de l'observateur. Au cours d'un examen radioscopique prolongé, la résistance électrique de l'ampoule doit diminuer à mesure que la sensibilité lumineuse de l'observateur devient plus grande. Mesurée par la longueur de *l'étincelle équivalente*, au moyen du *spintermètre*, le petit instrument que j'ai fait construire dans ce but, elle peut descendre de 9 centimètres environ à 3 centimètres et au-dessous.

Pour cette raison, les ampoules munies d'un régulateur de vide qui permet de faire varier à volonté, avec leur degré de vide, leur résistance électrique et par suite le pouvoir de pénétration des rayons qu'elles donnent, sont de beaucoup préférables aux autres. Rien ne vaut, à ce point de vue, les ampoules munies de l'osmo-régulateur imaginé par mon compatriote M. Villord.

Il n'est pas moins indispensable que l'ampoule, très facilement mobile, puisse être, au cours de l'examen radioscopique, placée par l'observateur en regard de tel ou tel point du thorax examiné. Cette condition est réalisée par le châssis porte-ampoule que j'ai fait construire dans ce but.

Mais il est utile surtout de pouvoir resserrer à volonté le faisceau divergent des rayons de Röntgen qui traversent le thorax de manière à restreindre la surface illuminée de l'écran ; c'est le moyen d'augmenter beaucoup, sur l'image radioscopique de la région plus particulièrement examinée, la précision des contours et la netteté des détails.

Dans ce but, j'ai fait construire un diaphragme de plomb, à ouverture variable, qui est une simplification du diaphragme-iris des microscopes. Ce diaphragme-iris, fixé au devant de l'ampoule et mobile avec elle, est complété par un petit instrument, l'indicateur d'incidence, qui montre à chaque instant, au cours de l'examen radioscopique, quelles que soient la position de l'ampoule et l'attitude du malade, le point de l'écran perpendiculairement frappé par les rayons de Röntgen : c'est au voisinage immédiat de ce point, dit *point d'incidence normale*, que l'image radioscopique se rapproche le plus, par la forme et les dimensions, de celles de l'organe correspondant.

C'est ainsi que, dans l'examen radioscopique d'un malade atteint ou soupçonné de tuberculose, on commence par prendre une vue d'ensemble des organes thoraciques en élargissant au maximum l'ouverture du diaphragme-

Digitized by Google

iris, puis on déplace l'ampoule de manière à ce que le rayon d'incidence normale traverse le sommet de la poitrine et on rétrécit l'ouverture du diaphragme-iris jusqu'à ce qu'on n'aperçoive plus sur l'écran que l'image des régions sus-claviculaires des deux poumons. Enfin on fait varier la résistance électrique de l'ampoule et par suite le pouvoir de pénétration des rayons qu'elle donne de manière à obtenir le plus grand contraste possible entre les parties claires et les parties sombres de l'image radioscopique.

Telles sont les conditions qui rendent plus précise et plus sûre la radioscopie appliquée au diagnostic de la tuberculose pulmonaire à ses débuts.

ON THE TECHNIQUE OF THE APPLICATION OF THE RÖNTGEN RAYS IN THE DIAGNOSIS OF TUBERCULOSIS.

By Dr. A. BÉCLÈRE,

Physician to the Hospital St. Antoine, Paris.

Professor Bouchard, my fellow-countryman and my teacher, was one of the first to gauge the value of Röntgen rays in the diagnosis of internal diseases. Less than a year after their discovery, on December 14, 1896, he read a paper before the Academy of Sciences, entitled "Röntgen Rays applied to the Diagnosis of Pulmonary Tuberculosis," in which he predicted the possibility of early diagnosis of this disease by means of the rays superseding all other methods.

I was asked to prepare a report on one of the subjects set down for discussion at the fourth Congress on Tuberculosis held in Paris in July, 1898, viz., the diagnosis of tuberculosis by means of Röntgen rays.*

If you will permit me to repeat some of the conclusions arrived at in that report, you will see, I think, that the interval that has elapsed has brought about little or no change:—

"No disease is more insidious at the outset, and in none do the lesions remain longer unobserved; either no functional trouble makes them suspected, or the pulmonary tubercles are so small and deeply seated that their presence is not detected by ordinary examination. Yet there is no disease in which it is more important to form an early diagnosis, for on this depends the success of the treatment.

"The Röntgen rays are particularly valuable in cases where tuberculosis is only suspected in the very early stage. Examination by the radioscope and radiograph supersedes all other methods and shows a diminution in the clearness of the lung at one of the apices, often accompanied by a diminished descent of the corresponding half of the diaphragm; and for a certain time this last symptom may be the only one observed. More often this new

* "Les rayons de Röntgen et le diagnostic de la tuberculose." Paris: J. B. Baillière et Fils. 1899. (Collection des Actualités Médicales.)

Digitized by Google

method confirms the record made on auscultation, and shows that attending the slight and dubious modifications of the respiratory murmur there is positive condensation of the pulmonary tissue. However, in other cases auscultation or percussion forestall Röntgen rays in proving change in the lung.

"As tuberculosis progresses and the symptoms become more marked, especially after bacilli are found in the expectoration, Röntgen rays are not so useful in diagnosis; on the other hand, this method of examination often indicates better than others the extent of the lesions, and thus becomes an important aid to prognosis. Finally, in the stage of excavation, it frequently, though not in all cases, shows the seat, number and size of the cavities which riddle the lung.

"In diseases which resemble tuberculosis the Röntgen rays help to arrive at a definite conclusion, either by showing that the lung is perfectly clear, or by revealing deep-seated lesions in the form of an interlobular encysted purulent mass, which accounts for consumptive symptoms."

If, on the one hand, the conclusions that I have referred to have scarcely changed in the three years, on the other hand the knowledge of the value of this mode of examination in pulmonary tuberculosis has spread more and more among doctors, and is, I think, universally recognised.

There are different ways of carrying out examination by Röntgen rays for purposes of diagnosis: examination with the radioscope, simple radiography, cinemato-radiography, and stereoscopic radioscopy and radiography. In this connection I should like to repeat what I said last year about this time at the International Congress of Electrology and Medical Radiology, held in Paris, in a report on Röntgen rays in the diagnosis of thoracic disease* :—

"These different methods, radioscopic examination, simple radiography, cinemato-radiography, and stereoscopic radiography, are of mutual assistance to one another in diagnosing thoracic affections, but to the medical practitioner they are not all of equal importance. Of the above methods, radioscopic examination is the simplest, easiest, quickest, and least expensive. But that is not all: it surpasses all the other methods in the amount and importance of the information it gives in a short time. Hence it comes first, and more often than not by its use in diagnosis it is possible to dispense with the others.

"The fluorescent screen shows the doctor, in a few moments, a multitude of different pictures in one region or in one organ. The picture varies with the general direction of the Röntgen rays, according as to whether the rays traverse the thorax directly from front to back, back to front, or transversely from right to left, from left to right, or obliquely in one of the intermediate diameters. Most often the patient stands up facing the tube or with his back to it. Under these circumstances the picture varies according to whether the tube, in the vertical frame in which it moves, is raised, lowered, or laterally displaced; in a word, according to the variable incidence of the Röntgen rays

* "Les rayons de Röntgen et le diagnostic des affections thoraciques non tuberculeuses." J. B. Baillière et Fils, Paris. 1901. (Collection des Actualités médicales.)

Digitized by Google

at each part of the thorax, at each point of the screen inclined in a different position. The picture varies again in its clearness and in the relative intensity of the different shadows of which it is formed, according to the power of penetration of the rays, which, within certain limits, can be modified at will in the course of examination. They are influenced by gravity, which modifies the relative position of the contents of the thorax, according to the position of the patient, erect or bent, immobile or suddenly moved. Finally, the picture changes most with the movements of the respiratory and circulatory organs, and to such an extent that it is possible to note and measure the digressions of the diaphragm, the changes in form and size of the lungs, heart, and aorta.

"Thus a few minutes suffice for a radioscopic examination and enable you to obtain from one thorax multiple images which complete and correct one another,—images which show not only the physical condition, but the most important organs carrying out their functions."

I can repeat to-day of the diagnosis of tuberculosis what I said a year ago with respect to thoracic affections in general—*the investigation of a case or a suspected case of tuberculosis should always be commenced by radioscopic examination.*

But to make the most of this new method of investigation, and particularly of radioscopic examination, experience has shown me that certain conditions must be taken into consideration.

These conditions, which I will lay before you, apply to the patient under examination, the observer, and the materials used.

A. Patient under examination.—His position should be such that it can be easily changed and that his respiratory movements are quite untrammelled. Hence, when possible, it is preferable to examine him standing up. If, on the other hand, this position is considered too tiring, he should be seated on a carefully selected seat, the height of which can be regulated and adjusted to all heights or figures, and which revolves to allow examination from every point. I find that a winding stool, like a piano stool, with a seat shaped like a bicycle saddle, is the best. It allows nearly complete extension of the legs on the pelvis, and thus enables the diaphragm to expand to its full extent. Finally, the patient may be made to lie down on a suspended linen sheet or a piece of wood, very permeable to Röntgen rays. But this position, sometimes indicated by the weak condition of the patient, hardly admits of much change of position, and so should be less commonly adopted than the other two.

B. The observer.—He should remain in the dark for a few minutes before beginning radioscopic examination, in order to acquire very great retinal sensibility to the light of the screen.

My own experimental observations * have proved by precise measurement that after ten minutes in the dark the sensibility of the retina to the light of the fluorescent screen has become fifty to a hundred times greater than it was

* "Étude physiologique de la vision dans l'examen radioscopique." 'Archives d'électricité médicale,' No. 82. 15 Octobre, 1899.

Digitized by Google

on emerging from broad daylight; it is about two hundred times greater after twenty minutes' observation, and after a longer interval it still increases.

C. Material used in investigation.—The increase in retinal sensibility after a period in the dark that I have just mentioned is of great practical importance from the point of view of the adoption of radioscopy among medical men. If it is preferable, as it undoubtedly is, for the generator of electrical energy, which feeds Crooke's tube, to have a good power, it is not absolutely indispensable for this power to be very great. As a matter of fact, a difference of one to two in the length and size of the spark which an induction coil or a static machine can supply, matters little, whereas a stay in the dark increases the retinal sensibility of the observer to the extent of a hundred and two hundredfold. I have described * and frequently use a particular model of statical machine by Wimshurst, with four glass cylinders, moved by hand, and small enough in size to take to patients' houses. This simple little machine, which is the radiogenic apparatus *par excellence* of the provincial medical man, is all that is required to give an excellent view of the thoracic organs on the fluorescent screen, to eyes which have been suitably prepared by an interval of darkness.

I now come to a very important point on which I particularly desire to lay stress. As you know, there are any number of Röntgen rays, which differ from one another in their unequal power of penetration, and the same tube may produce rays more or less penetrating, according to its degree of electrical resistance.

The penetrating power of the rays furnished by the tube evidently must be increased according to the thickness and density of the organs examined; thus it must be greater in order to examine a man's thorax, with his strong muscles, than it need be for a thin woman's thorax, if as clear a picture is to be shown on the fluorescent screen of one as of the other.

In practising radioscopy I have found out another condition, less apparent at first sight, but quite as necessary as the preceding one to the clearness and accuracy of the pictures thrown on the screen. From my experience *it is indispensable* that the power of penetration of the Röntgen rays should, all things being equal, be exactly suited to the luminous sensibility of the observer. To be more precise, in the course of prolonged radioscopic examination carried out on a succession of patients, as in hospital practice, the electrical resistance of the tube, and consequently the power of penetration of the rays that it produces, must, all things being equal, diminish as the luminous sensibility of the observer increases.

We are able, as you know, to measure with a fair amount of precision the electrical resistance of the tube; this is done by slowly drawing together the two conducting wires which bind the tube to the induction coil or to the statical machine till a spark flies up between the two. This occurs at the precise moment when the electrical resistance of the stratum of air which

* "Sur une petite machine statique propre à l'examen radioscopique au domicile des malades." 'Archives d'électricité médicale.' 1900.

Digitized by Google

separates the two conducting wires no longer exceeds that of the tube; it is also called the spark which is equivalent to the resistance of the tube, or, more briefly, the equivalent spark. I have had a very simple little instrument made that I have called the spintermeter,* which is of great daily service to me in measuring exactly the length of this equivalent spark and in defining the penetrating power of the rays given by the tube. Now I will tell you what I have proved many times at the Hôpital St. Antoine, by the help of this instrument, when the radioscopic examination exceeds half an hour.

Quite at the beginning of the examination, if I wish to obtain clear pictures on the screen, I see that the electrical resistance of the tube is measured on the spintermeter by a spark at least ten centimeters long. But as the examination progresses and my luminous sensibility grows, I have to diminish the electrical resistance of the tube until the spintermeter only registers a spark of three centimeters long at the outside.

From the foregoing it will be seen that it is absolutely necessary in the course of radioscopic examination to be able to vary at will the amount of vacuum in the tube; it is the best way of varying at the same time its electrical resistance and the power of penetration of the rays that it gives. In other words, it is necessary to use a tube provided with a space regulator. The best tube of that kind that I know is the one provided with an osmometer, invented by a distinguished compatriot of my own—M. Villard—whose valuable works on Cathode rays you are acquainted with. The principle of Villard's osmometer depends, as you know, on a peculiarity of platinum discovered thirty years ago by two other countrymen of mine—Messieurs Troost and Sainte Claire-Deville—its permeability to hydrogen, and to hydrogen only, when red-hot. Villard's osmometer is a small platinum tube fastened to the tube communicating with the air of the tube at its open end and closed at its free end. All that is required is to make it red-hot in the flame of a spirit-lamp or a gas-burner, in order that the hydrogen of the flame may traverse the side of the platinum tube which has become porous, and increase the pressure inside the tube. Take the platinum tube out of the flame and it cools down, ceases to be permeable to hydrogen, and the gas which has got into the tube cannot escape again. But by means of M. Villard's very ingenious invention it can be dispelled at will; time, however, is too short for me to describe it now.

Thus the degree of vacuum in the interior of tubes provided with Villard's osmometers can be diminished or increased at will; at first they were only made in France, but now I know similar ones are made in England and Germany. These are the tubes I recommend, in preference to other kinds, for radioscopic examination of the lungs, particularly in the early diagnosis of tuberculosis, on account of the ease and certainty with which the power of penetration of the rays is varied at will.

Another important matter in radioscopic examination is that the tube

* "La mesure indirecte du pouvoir de pénétration des rayons de Röntgen, à l'aide du spintermètre." 'Archives d'électricité médicale,' No. 88. 15 avril 1900.

Digitized by Google

should be quite freely movable, and for this purpose a support should be used similar to that invented several years ago by Dr. Guilleminot. I had a support of the kind made, which I described in the *Archives d'électricité médicale*,* and which was primarily intended for radioscopic examination in an upright posture, seated or semi-recumbent, but which, in addition, can be used for simple radiography or stereoscopy of all parts of the body on a patient lying horizontally in the dorsal, abdominal, or lateral position. I use this support most frequently when I examine a patient in an upright posture or seated. It has the great advantage of being able to raise or lower the tube very easily in a vertical direction, not only according to the height of the patient under examination, but according as one wishes to explore this or that portion of the thoracic organs.

A third condition, no less important than its predecessors to good radioscopic examination of the lungs, especially in the examination of the apices in patients only suspected of tuberculosis, is to be able to focus at will the diverging bundle of Röntgen rays, at the exit of the tube, which traverse the thorax, in order to limit the illuminated surface of the fluorescent screen; in this way the precision of the outlines and the clearness of the details in the radioscopic picture of the region more particularly examined are increased.

The use of a lead diaphragm has been recommended for some time with this object. Instead of having lead diaphragms of different sizes it is more convenient to have one that can be regulated. Therefore, I had a lead diaphragm made which is a simplified reproduction of the iris-diaphragm of the microscope. The aperture is square and the centre is fixed, whilst the side can be increased or diminished at the will of the operator. Not only can the size of the opening of the iris-diaphragm be varied at will, but its shape even can be varied, and instead of being square it may become a narrow rectangular band, when, *e.g.*, one wishes to compare in one patient two symmetrical regions, such as the two halves of the diaphragm or the summits of the two lungs.†

This iris-diaphragm fixed in front of the tube, and movable with it, is completed by a little instrument, composed of two strands of thread crossed and called the *incidental-indicator*, which shows at any moment during the course of the radioscopic examination, whatever the position of the tube and the attitude of the patient, the point on the screen on which the Röntgen rays are thrown perpendicularly. This point, called the point of normal incidence, is very important to recognise, because in its neighbourhood the form and dimensions of the radioscopic picture differ least from those of the organ that this picture represents.‡

Therefore in radioscopic examination of a case of tuberculosis, or of suspected tuberculosis, the first thing to do is to dilate the opening of the iris-

* "Les instruments auxiliaires de l'emploi médical des rayons de Röntgen." 'Archives d'électricité médicale.' 15 juin 1901.

† "L'emploi du diaphragme-iris en radioscopie et son utilité pour la détermination du point d'incidence normale." 'Archives d'électricité médicale.' No. 94. 15 octobre 1900.

‡ "Les instruments auxiliaires de l'emploi médical des rayons de Röntgen." 'Archives d'électricité médicale.' 15 juin 1901.

Digitized by Google

diaphragm to the utmost, in order to obtain a comprehensive view of the thoracic organs, and to raise the tube to such a height that the rays of normal incidence pass as nearly as possible by the base of the heart. After this general view, the different layers of the lungs must be looked at in succession, by raising or lowering the tube, so that the normal rays of incidence traverse the part specially under examination. At the same time the opening of the iris-diaphragm should be contracted so as to only show on the screen the picture of the region specially explored. For example, in examination of the apex of the lungs, the tube is so raised that the rays of normal incidence pass slightly above the clavicles, and the opening of the iris-diaphragm is contracted till the screen only shows the picture of the supraclavicular region of the two lungs. Finally, either by heating Villard's osmometer or by heating the tube itself, the electrical resistance of the latter can be varied, and consequently the power of penetration of the rays it produces, so as to obtain the greatest contrast possible between the clear and the dark parts of the radioscopic picture.

Pray forgive my having taken up so much of your time with technical details; my justification is the importance of these details in the definite diagnosis of pulmonary tuberculosis by means of radioscopic examination. Nothing should be left undone that can assist in early diagnosis of this malady, for on its early recognition the success of treatment depends.

DR. JAIME R. COSTA, *Professor in the Faculty of Medicine, Buenos Aires.*

J'AI eu l'occasion, à Paris, de suivre pendant quelque temps le service de radiographie clinique que dirige M. le Dr. Béclère, à l'hôpital St Antoine, et je puis affirmer,—ayant eu aussi l'opportunité de visiter d'autres installations, —que dans aucune on arrive à acquérir les résultats qu'au point de vue du diagnostic j'ai vu obtenir à M. le Dr. Béclère. Moi-même j'ai travaillé pendant trois ans à la chaire de Physique médicale que j'occupe à Buenos Aires, faisant beaucoup d'examens radioscopiques, et je déclare que j'ai été surpris des résultats que dans l'examen du thorax on obtenait à l'hôpital St Antoine.

J'attribue ces résultats aux perfectionnements introduits par M. le Dr. Béclère, surtout à son diaphragme-iris et à la mobilité qu'il arrive à donner au tube producteur des rayons Röntgen, de manière à l'avoir toujours dans l'axe de son incidence normale.

On voit très bien les différences d'opacité présentées par les sommets pulmonaires, si importantes pour le diagnostic de la tuberculose commençante,—on peut attribuer aussi son vraie origine à un épanchement pleural, dont on méconnaît l'étiologie, on peut faire très bien le diagnostic différentiel entre les adénopathies du médiastin et les aneurismes de l'aorte, surtout si on a recours à l'examen oblique du thorax, qui délimite très bien les contours de cette artère. J'attribue aussi une grande valeur au méthode de M. Béclère—

Digitized by Google

qui adapte la résistance de son tube à l'épaisseur du thorax et surtout à la sensibilité retinienne au moment de l'examen, sensibilité qu'il peut mesurer par l'étincelle équivalente—et à la grande mobilité qu'il donne à son malade.

Je crois qu'un examen du thorax fait suivant ces principes acquiert une valeur considérable, et ces résultats peuvent se comparer à ceux que donnent l'auscultation et la percussion, qu'il vient de compléter, et que dans certains cas il décèle des lésions que les autres méthodes ne pouvaient faire soupçonner.

Je termine en exprimant mes vœux pour que la radioscopie soit usitée régulièrement, suivant les précieuses indications données par M. le Dr. Béclère dans tous les services où l'on soigne des tuberculeux ; ces données sont si instructives qu'ils compenseront amplement les frais et les fatigues de l'apprentissage.

SUR L'EMPLOI DES RAYONS DE ROENTGEN POUR LE DIAGNOSTIC PRÉCOCE DE LA TUBERCULOSE PULMONAIRE.

PAR M. LE DR. BONNET LEON, *Paris*.

MONSIEUR LE PRÉSIDENT, MESDAMES, MESSIEURS,—J'ai l'honneur de communiquer au Congrès la note suivante "sur l'emploi des rayons de Roentgen pour le diagnostic de la tuberculose pulmonaire, et particulièrement pour le diagnostic de la *période prétuberculeuse*."

Mes conclusions sont basées sur plus de 600 cas que j'ai personnellement observés de 1897 à ce jour, soit dans ma clientèle privée, soit à mes cours libres de la Faculté de Médicine, soit aux Dispensaires anti-tuberculeux de la rue St Lazare et de la rue Marcadet, à Montmartre.

Ces 600 cas, choisis dans un nombre considérable d'autres, ont été soumis à l'auscultation préalable par plusieurs confrères, MM. les Drs. Aubert, Bondon, Coste de Lagrave et Parent.

Je les ai ensuite examinés à l'écran fluorescent, sans connaître, bien entendu, le résultat de l'auscultation.

Les rayons X se sont trouvés en défaut 1 ou 2 fois sur 100 ; notamment chez les enfants et les femmes ils n'ont pas toujours signalé de lésions tuberculeuses assez nettes à l'auscultation.

Par contre, dans 30 cas sur 100, ils ont précisé les lésions, montré des zones d'opacité plus considérables que la percussion ne les avait déterminées et assez souvent (5 fois sur 100), ils ont même permis de porter des diagnostics au premier degré qui avaient échappé aux autres méthodes d'examen.

L'usage courant des rayons de Roentgen, de nombreux auteurs l'ont prouvé, est donc égal, sinon supérieur à l'emploi des anciens procédés pour le diagnostic des trois périodes de la tuberculose ordinaire.

Je me contenterai, à ce propos, d'apporter par les nombreux cas dont je viens de parler, une contribution aux études déjà faites.

J'ajouterai simplement que mon expérience me fait voir dans la radioscopie

Digitized by Google

une méthode facile et surtout rapide d'examen du thorax, à recommander dans un milieu encombré et bruyant comme celui d'un dispensaire, d'un régiment, d'un conseil de révision.

Mais je désire appeler spécialement votre attention sur un *moyen particulier de diagnostic* par les rayons X, à employer dans cette période de la maladie qu'on a nommée *prétuberculeuse.*

A ce degré du mal, ni la percussion ni l'auscultation les plus attentives ne peuvent déceler une modification dans la sonorité ou dans le murmure vésiculaire; le microscope ne trouve pas ici son emploi, puisqu'il n'y a pas de crachat bacillaire, le tubercule étant encore fermé; l'examen à l'écran ne montre pas d'opacité, qui n'existe pas ou qui est masquée par de l'emphysème; la plaque photographique peut même, malgré sa très grande sensibilité, ne pas enregistrer de lésions.

Beaucoup de personnes, soumises à ces examens négatifs, avec une santé relativement bonne, sont cependant en puissance de tuberculose et sous la menace du mal qui montre quelques mois après des signes non douteux à la *radioscopie ordinaire* ou à l'auscultation.

Un mode spécial d'application des rayons X permet de s'en assurer et de porter le diagnostic si incertain de prétuberculose.

Il s'agit d'observer, a *l'aide de l'écran*, les mouvements respiratoires et très attentivement *le jeu du diaphragme.*

Depuis que je m'occupe de la méthode Roentgen, j'ai contracté l'habitude d'examiner au fluoroscope toutes les personnes saines ou malades pour ainsi dire des pieds jusqu'à la tête, en insistant sur le thorax, la partie du corps la plus visible à l'écran.

Je fus très fréquemment frappé par le mauvais fonctionnement du diaphragme, le plus puissant des muscles inspirateurs.

J'en cherchai la cause, sans la trouver le plus souvent, et j'attribuai ce vice de fonctionnement à une mauvaise habitude respiratoire ou à une particularité individuelle.

J'avais remarqué, et de nombreux confrères avaient publié, que les tuberculeux respiraient mal, en général, et particulièrement chez eux que le jeu du diaphragme et l'expansion du thorax étaient diminués du côté atteint; mais chez beaucoup de mes sujets examinés, dont l'état de santé apparente était d'ailleurs satisfaisant, je ne pus découvrir à l'écran ou à l'auscultation la moindre trace d'opacité ou de lésion expliquant cette insuffisance des mouvements diaphragmatiques.

Quelques-uns étaient cependant atteints d'affaiblissement général, de dyspepsie, de neurasthénie, mais rien d'évident et de net ne pouvait expliquer ce que je trouvais: c'était, tantôt la diminution notable de l'amplitude des excursions diaphragmatiques qui, chez l'homme sain, vont de la 6e à la 8e côte et mesurent facilement de 8 à 10 centimètres, tantôt l'irrégularité de jeu d'un des côtés du muscle ou du muscle tout entier, tantôt l'anomalie de sa courbure.

Mais, ayant eu l'occasion de revoir et, examiner une deuxième fois quelques-unes de ces personnes, à six ou dix-huit mois d'intervalle, je fus à même de

Digitized by Google

constater chez plusieurs d'entre elles des lésions tuberculeuses débutantes ou assez avancées et correspondant à l'irrégularité remarquée.

Il se présenta une dizaine de cas analogues en assez peu de temps.

En continuant ces recherches et en provoquant même les examens chez une série de personnes saines ou à peine suspectes, j'arrivai à dresser une liste assez longue d'observations typiques. J'ai pu sur 600 cas en réunir une centaine qui me paraissent très concluants.

Ce nouveau signe de diagnostic prétuberculeux que j'appellerai *signe diaphragmatique* est assez simple à reconnaître.

Tout médecin familiarisé avec l'examen radioscopique du thorax, connaissant le fonctionnement normal des muscles inspirateurs et l'aspect ordinaire du diaphragme pendant les deux temps de la respiration, sera frappé immédiatement par une anomalie dans ce fonctionnement ou cet aspect; il pourra sans difficulté en apprécier le degré, vérifier l'éclairement des poumons, contrôler l'expansion de la cage thoracique.

En l'absence de cause évidente, comme par exemple celle d'une tumeur solide ou liquide du thorax ou du bassin, celle d'inflammation d'organes voisins, de manifestations rhumatismales, de maladie du système nerveux, d'intoxication saturnine, de pneumonie et congestion pulmonaire, il devra songer à la tuberculose commençante et surveiller le sujet, chez lequel quelque temps après il trouvera 90 fois sur 100 des lésions manifestes de tuberculose.

Ces anomalies du jeu du diaphragme doivent vraisemblablement résulter :

1°—ou de pleurésies le plus souvent méconnues et n'ayant laissé comme traces que des adhérences légères ;

2°—ou bien d'altérations bacillaires imperceptibles du tissu pulmonaire dont l'élasticité et la densité sont modifiées ;

3°—ou bien encore d'une inertie musculaire provoquée par la toxine issue de tubercules disséminés.

Cette dernière hypothèse doit être fréquemment exacte, si l'on songe, par exemple, que l'intoxication par le plomb paralyse le diaphragme et que les toxines de plusieurs maladies infectieuses occasionnent diverses paralysies très nettes. D'ailleurs dans la tuberculose pulmonaire, il est indiscutable que tous les muscles et surtout ceux du tronc et des membres supérieurs sont atteints de parésie douloureuse.

Or, de tous les muscles, le diaphragme est le seul dont la conformation et la situation permettent de voir les mouvements par l'écran radioscopique.

M. le Dr. Williams et M. le Dr. Béclère ont déjà signalé l'abaissement moindre du diaphragme correspondant au côté d'une lésion pulmonaire ou tuberculeuse, en décrivant cette diminution de l'abaissement comme *contemporaine de l'altération déjà visible* du poumon.

M'appuyant sur de nombreuses observations, j'estime que, *en dehors* de toute *altération visible du poumon*, une anomalie respiratoire *quelconque* dans le jeu du diaphragme principalement, doit, lorsqu'elle est constatée à l'écran, toujours éveiller l'idée de tuberculisation.

Un schéma de diverses anomalies est annexé au plan du Dispensaire de Montmartre, exposé par M. le Professeur Landouzy au Musée du Congrès.

Digitized by Google

Peut-être dira-t-on qu'une respiration faible, mauvaise, irrégulière est plutôt une prédisposition à la tuberculose qu'un signe de l'éclosion tuberculeuse. La distinction serait subtile et au point de vue pratique de peu d'importance, puisque dans l'un et l'autre cas le traitement s'impose.

Il convient cependant de faire observer que la tuberculose peut envahir des individus dont la respiration est excellente et que dans ce cas, dès le début, elle rend cette respiration défectueuse.

Hutchinson a montré depuis longtemps par l'emploi du spiromètre qu'au début de la phtisie, la capacité inspiratrice diminue de plus de 1/10.

Nous pouvons donc admettre qu'il y a entre la tuberculose et le signe diaphragmatique une relation de cause à effet.

Il est facile, ajoutons le encore, de constater à l'écran que les enfants et les adultes issus de parents phtisiques présentent presque toujours le signe diaphragmatique, comme s'ils étaient en puissance du poison tuberculeux.

En atteignant les fonctions respiratoires et surtout celles du diaphragme, qui sont les plus importantes, la tuberculose prépare ainsi un terrain très favorable à son rapide développement.

Peter avait établi "qu'à un minimum de fonctionnement correspond un maximum de tuberculisation."

En effet le jeu normal des poumons, de nombreux faits le démontrent, prévient et enraye la marche du bacille.

La *respiration profonde* s'oppose à la congestion, à l'invasion morbide, empêche la diminution du poumon, dilate en tous sens le thorax, amène la résorption des indurations, détruit les adhérences diaphragmatiques, chasse des alvéoles l'air vicié, fait pénétrer l'oxygène dans les sommets, qui en sont insuffisamment pourvus, et développe l'emphysème curateur autour des lésions.

Il faut en conséquence rechercher, au moins chez tous ceux qui sont prédisposés par l'hérédité, par le milieu, par l'alcoolisme, les signes que nous offre l'emploi de la radioscopie, et opposer au trouble fonctionnel, outre le traitement général, les exercises de gymnastique respiratoire applicables à chaque cas particulier.

Le signe *diaphragmatique*, très visible, peut même être *mesuré;* il n'est pas contestable ou relatif comme ceux de l'auscultation. C'est un signe important pour diagnostiquer le début de la tuberculose, à une période où le mal est facilement et sûrement curable.

Digitized by Google

THE IMPORTANCE OF THE X RAYS AS A MEANS FOR THE EARLY DIAGNOSIS OF TUBERCULOSIS.

By Dr. ESPINA Y CAPO,

Of the Royal Medical Academy of Madrid; Professor of Tuberculosis in the Provincial Hospital of Madrid, Inspector of Public Health, etc., etc.

Mr. President and Gentlemen,—I must be very brief, because I am in the city where "time is money," and furthermore because my purpose is solely to obtain for radiography and radioscopy their free use as means of diagnosis in tuberculosis.

My work comprises three parts: firstly, radioscopy; secondly, radiography; and thirdly, the technique of both in relation to tuberculosis. In the first part I shall speak of radioscopy in cases of sound and tuberculous lungs, in the second of the use of radiography in the different stages of the disease, whilst in the technical section I shall refer to the best method of obtaining the best results in both.

Radioscopy.—Radioscopy as a means of diagnosis in tuberculosis has constituted, since its appearance in the clinical field, one of the means in which the greatest hopes have been placed, so much so that since the fourth Congress on Tuberculosis held in Paris it has been considered the equal both of percussion, superficial and profound, and of auscultation.

In that fourth Congress, however, we could not speak so affirmatively, because we were not yet in possession of positive instances which would leave no room for doubt. To-day it is already impossible to do without this potent means, adopted in all countries and by all practitioners.

Partisan as I am of the parasitic doctrine, I will not treat of the radiographic diagnosis of open pulmonary tuberculosis, because the analysis of sputum, and the presence in it of the bacillus of Koch, affirm so positively the existence of tuberculosis as to satisfy every physician. Radioscopy, in the case of open tuberculosis, serves only as a potent means of settling the topography of the lesions, and although this is very important, it does not deserve to fix our attention, because we prefer to employ our time studying the true use of the X rays in the diagnosis of pulmonary tuberculosis in its first period full of doubts, and in which therapeutical intervention, being more rapid and convenient, is also more efficient.

There is another question of transcendent importance, in which the X rays have assisted our investigation with such clearness that this peculiar aspect of the question justifies our fixing it in our attention. I refer to glandular tuberculosis, the commencement, in many cases, of pulmonary tuberculosis, and in all the inseparable companion of the disease.

Apart from the data given by all authors in explanation of the changes of colour from which the radioscopic image of the lungs suffers in the incipient, stationary, and cavernous stages, we shall not refer to them, because at this

Digitized by Google

Congress we are obliged to present subjects of some novelty. I am going to mention, however, two circumstances, which, I believe, perhaps through ignorance, possess that peculiarity, and which we can only observe, especially one of them, by radioscopy.

Radioscopy constitutes a powerful means of studying the dynamic forces. It is not possible to see by radiography all the functions and all the movements that characterise each function. For this reason the normal physiology of, for example, a joint during the different phenomena which constitute movement and action, can alone be seen on the fluoroscopic screen, an impossibility with the radiographic plate.

The two phenomena to which we are about to refer appertain to actions of organs in a state of movement, and for that reason can only be observed by radioscopy. The first is the heart. Ever since Wells drew attention to the diagnostic value of tachycardia, all specialists, and those who devote themselves specially to the study of tuberculosis, have set great store upon this point, raising it to an important position in the early diagnosis of tuberculosis. One of the most efficient means of verifying this datum is by fluoroscopy. As we never lose sight of this diagnostical resource in our department of tuberculosis, we can affirm, in an indubitable manner, that, with the fluoroscopic screen, the datum of Wells is seen in more than 94 per cent. of cases of tuberculosis, with so much clearness as to remove all doubt on this subject. I shall, moreover, refer to a sign which, I think, has not been touched upon by other authors—namely, that this tachycardia is not rhythmical.

There is another sign on the heart of the tuberculous, but we shall study it when we treat of radiography.

The second example that may be taken for radioscopy, being essentially functional, and one which we have not seen referred to by any other author, appears to our observation with as much, or even more, constancy than tachycardia. We refer to the visibility of the diaphragmatic excursion, both in inspiration and expiration, a phenomenon that, as is easily understood, can be appreciated neither by common means of exploration nor by radiography.

This phenomenon, which under normal conditions is rhythmical and symmetrical on both sides, in tuberculosis is neither rhythmical nor symmetrical. We have observed that in the tuberculous the diaphragm does not rise to the same height on the diseased as on the healthy side, and that when the two lungs are affected the ascension is shorter on the worse side; in similar manner in the case of a tuberculous patient whose diaphragm on one side has no ascension, or where it is very short, we deduce from this the correspondingly greater impermeability of the lungs.

Radiography.—Accepting also, in the case of radiography of tuberculous lungs, as much as we have related respecting the several degrees of transparency and depth of shadows in the first stages, darkness in the tract of the ganglionic and lymphatic vessels, opacity in the congested lung, and very great transparency of the caverns, we shall only say with regard to radiography that

Digitized by Google

it is possible with it to discover the data, also of a certain novelty, in the radiogram of the tuberculous.

In our remarks on tachycardia when dealing with radiography, we said that the heart gives a sign easier of record by radiography than radioscopy. Since the adoption of this valuable means of diagnosis, the small size of the tuberculous heart has attracted our attention, being a true cardiac atrophy. If this, as we believe, is a positive indication of tuberculosis, we shall arrive at a definitive early diagnosis with greater ease if we find in our radioscopic and radiographic explorations small heart, especially if the investigations are accompanied by tachycardia: moreover, we have an explanation of the relations existing between the smallness of the heart, aortic narrowness, and the frequency of tuberculosis in such subjects, which determine the rule existing among these phenomena.

Another datum that may be observed is the peculiar shape of the intercostal spaces. In other works relative to the same matter, we have referred to the difficulty of performing percussion on tuberculous patients.

The causes of this difficulty are two: the first the extreme narrowness of the intercostal spaces, it being impossible to place the fingers between the ribs, and the second the conical form of the tuberculous thorax. Now before the appearance of the Röntgen method we could hardly understand these difficulties, but since the clinical adoption of this new system, the reasons become apparent. In the radioscopic image is observed a disposition of the ribs strongly resembling the peculiar arrangement of roof tiles, in such a manner that the inferior rib almost touches the anterior face of the inferior rib, the intercostal space being so small, particularly from the fourth rib downwards, that it is difficult to place the finger upon which percussion is made. With radioscopy this disposition of the ribs is very clear, and functional phenomenon corresponding to the diminution of the respiratory excursion in the tuberculous, impossible of diagnosis in any other manner, is very evident.

As we see, radiography permits us to obtain information of transcendental importance in the living, in the functional, in the active, and for this reason it is not possible to dispense with this means of diagnosis of tuberculosis.

The transient nature of the radioscopic image, and the need of a permanent image, obliges us to supplement radioscopy with radiography. It is true that radiography cannot give us any information as to function, but instead, with this second method, anatomical facts are permanently recorded, as in a book ever open to study.

Amongst the most interesting facts that we can select from our experience in radiography are the deformities of the bones characteristic of tuberculous thorax. There are in the thorax certain bones, which for their bearing on respiration and also from the muscles that originate from them, are organs of transcendental importance in the osseous respiratory apparatus. They are the clavicle and the scapula. Their position, form and size also produce very important modification in tuberculosis.

In taking a radiograph of a tuberculous subject, we see the modifications

Digitized by Google

of these bones; the clavicle principally presents an augmentation on the inflection of its external extremity, and resembling a hyperplasia, but without being sufficient to constitute an alteration of the tuberculous epiphysis, takes the shape of the head of a mace. The scapulas are higher, and the internal and superior end is more elevated, swinging towards the centre the point and internal edge which are raised on account of the peculiar disposition of the ribs and spinal column.

Without entering into the pathological physiology of these facts, which entirely depend upon the great respiratory necessities of the tuberculous, united with the reduction of their respiratory field, we must remark that radiography shows these pathological heteromorphies with great clearness.

The roof-tile disposition of the ribs, of which we have spoken before in speaking of radioscopy, can be seen in an evident and clear manner by radiography.

Besides these deformities of the skeleton, radiography also shows us the small size of the heart. Respecting this organ radiography has achieved wonders, not alone in tuberculosis, but as to its real situation; but putting aside this latter fact, and occupying ourselves solely with those data which we can recognise in tuberculosis by the heart, we shall mention two that have, we think, a certain novelty and importance.

The first is the greater extension of the heart towards the lines of Traube and Friedrich on the right side, this fact showing, in our opinion, a compensatory insufficiency of the tricuspid valve, and, for that reason, ventricular hypertrophy on the right side; the radiographic image of the heart in tuberculous persons thus acquires a strong resemblance to the image of the tired heart of the emphysematous.

The second datum refers to the special form of the heart of the tuberculous, similar to the foot of a Chinese woman in its left ventricular border, remembering the growth of the deformed foot of the Chinese.

Respecting the lungs the facts of the first, second and third periods are so classic that we do not consider them worthy to occupy your attention: for our part they are simply confirmatory of what we related in the fourth Congress held in Paris.

Technique of Radioscopy.—To observe the wonderful phenomena of radioscopy under the best conditions of visibility, complete darkness is necessary, suppressing it until the light is produced by the Röntgen tube: for this purpose we have had a special box constructed, to conceal the light completely, provided with a simple mechanism adaptable to any exploration that may be necessary.

Another trouble that must be got over is the difficulty of applying the fluoroscopic screen, which is commonly rigid and mounted in a frame. In this form the screen can, in tuberculous cases, only be applied upon the sternum or upon the internal edge of the scapula. It is known that in radiographic images size and intensity depend upon the distance between the part under observation and the surface of the screen, so that with rigid screens it is not possible to obtain a corresponding equality of definition and

Digitized by Google

intensity over all the surface, and to obtain such a result it is necessary to use a flexible screen mounted on card-board in such a manner that it can be adapted to the convexities of the thorax. One of our colleagues, a very competent person in these matters, Doctor Redondo, Fleet Surgeon of the Spanish Navy, advises the employment in radioscopy, as a radioscopic screen, of the strengthening screen whose image resembles in depth of tone the radiographic image, and gives as fine detail as the ordinary fluoroscopic screen.

This application of the strengthening screen makes the solution of the problem easy, because it avoids having to change in the laboratory the fluoroscopic material, of which, as we have said before, the rigid screen is constructed.

This last modification of the fluoroscopic technique is relative to the examination of patients that must have fasted previously, to obtain with more clearness the semi-lunar space of Traube, and to observe with greater ease the phenomenon of the diaphragmatic excursion previously referred to.

Radiographical Technique.—For some time we have omitted as much as possible the use of strengthening screens. This is not the moment to analyse in detail their advantage or inconvenience. I will refer, however, to one amongst its inconveniences which, I think, is pertinent at this moment—*viz.*, the turbid and disseminated nature of the image. When, therefore, we treat of images that require great detail, obtained in subjects capable of enduring the exposure—a very short time—that is required to-day to obtain a radiograph, we prefer to work without a strengthening screen in order to obtain a completely clean image, with richness ot detail, especially in pleurisies and patients with but small caverns.

Another interesting subject in radiographical technique, that also gives clearness to the image, and richness of details, is the distance that should exist between the tube and the plate. Without discussing theory, but confining ourselves to the collected facts, we have succeeded in fixing this distance at 50 centimetres from the thorax, at which we obtain the maximum richness of detail.

The last matter that will occupy us in this address refers to the development of radiographic plates. We have tried nearly all the old and new developers, from the classic iron and pyrogallic acid to diamido-phenol and diamido-resorcine, and from this study we have deduced the necessity of never developing in radiography with old liquids in any one proportion, as much with the developing as with the fixing agents; we use the diamido-phenol ($\frac{1}{2}$ per cent.), with crystallised sulphite of soda (6 per cent.) as a developer, and Enckel's (Swiss) hyposulphite (25 per cent.). With these products we make our baths instantly, that is for two or three radiographs only, especially in Madrid, where the purity of the Lozoya's solution (4° hydrometric) permits of its employment in all photographic laboratories.

We finish, as we began, conscious of having said nothing either good or new; but the call of duty has bade us add our feeble efforts to this Congress in which every altruism has its place in the fight with this dreadful tuberculosis.

Digitized by Google

DIFFICULTÉ DE L'INTERPRÉTATION DES IMAGES EN RADIOLOGIE.

PAR M. LE DR. MIGNON, *de Nice.*

BIEN que l'emploi des rayons X soit d'une utilité incontestable, beaucoup de praticiens, dans le cas où il serait utile, n'y ont pas recours. Ce manque de confiance tient à ce que souvent des examens ont été pratiqués par des gens incompétents et ont donné par conséquent des résultats erronés.

L'interprétation des images est toujours difficile, même dans l'inspection du poumon, qui est pourtant l'un des organes les plus faciles à examiner. On sait que la tuberculose produit en se développant une teinte sombre variable suivant les lésions. Sans insister sur ce point, je désire attirer l'attention sur l'aspect du poumon chez les tuberculeux guéris, ou du moins considérés comme tels au point de vue clinique. Si le malade a eu des petites lésions, elles passent inaperçues ; au cas contraire, elles donnent des zônes sombres assez intenses et en général mieux limités que dans les cas de tuberculose en cours d'évolution. Ceci pourrait s'expliquer en considérant que le tissu fibreux ou crétacé de la guérison est très différent par sa densité du tissu sain, tandis que les lésions en voie de développement sont souvent entourées d'une zône congestive, qui empêche de différencier aussi nettement leur limite.

S'il est souvent difficile pour nous médecins d'arriver par l'emploi des rayons X à une opinion précise, je me demande quel avis peuvent donner les industriels auxquels le public s'adresse volontiers depuis la découverte de Röntgen.

Leur ignorance de l'anatomie et de la pathologie leur enlève toute compétence ; et d'autre part un médecin non exercé à ce genre d'examen ne peut tirer profit de leurs indications. Mieux vaut ne pas avoir recours aux rayons X que de les mal employer ; car ils deviennent alors une cause d'erreur et la méthode en est dépréciée. Un radiologue doit être à la fois électricien et médecin, pour prétendre, après avoir acquis les connaissances nécessaires, donner un avis autorisé. En dehors des erreurs auxquelles, dans le cas contraire, est exposé le malade, il court aussi certains dangers que le médecin évitera plus facilement qu'un autre expérimentateur en surveillant et en interrogeant son malade.

Puisque un examen aux rayons X a pour but un diagnostic difficile, et que cet examen lui-même peut présenter des dangers non moindres que l'emploi de certains traitements, il me semblerait logique de considérer comme exercice illégal de la médecine le fait de se livrer à ces examens ; les malades doivent être protégés du double danger que leur fait courir leur ignorance, celui d'être presque sûrement trompés et souvent estropiés.

Digitized by Google

DEMONSTRATION ON CASES OF LUPUS VULGARIS TREATED BY FINSEN'S METHOD AND BY THE X RAYS; ALSO RESULTS OF OTHER FORMS OF TREATMENT.

DR. SEQUEIRA showed a series of nine cases treated by him at the London Hospital. The patients had suffered from lupus vulgaris for from three to ten years. Five of them had been treated by concentrated chemical light only (Finsen's method), three had been treated by the X rays only, and one had been subjected first to the X rays and then treated by Finsen's method.

The essential conditions for success in the Finsen treatment are:—

(1) The application of a sufficiently intense light, concentrated sunlight, or electric arc light.

(2) The use of an apparatus to compress the skin and render it anæmic, and thus permit the penetration of the chemical rays; and

(3) The elimination of the effects of heat by making the compression apparatus hollow and passing a current of cold water through it.

The treatment is painless. A reaction sets in from six to twelve hours after the application, redness, swelling and frequently vesication occur.

The cases illustrated the following points:—

(1) The complete disappearance of the lupus nodules.

(2) The smooth, soft, supple cicatrix, which in some instances was almost indistinguishable from normal skin.

(3) In one case, the disappearance of a keloid occurring in the scar left by previous scraping.

The drawbacks to the Finsen method are the length of time necessary for efficient treatment. One of the patients shown had had over two hundred sittings, others had had one hundred, and one had had only twelve. The expense of the treatment was an important matter, and unfortunately it was impossible, except in rare cases, to treat lesions of the mucous membranes.

The X ray treatment was carried out in the manner described by Schiff. It is painless, and with hard tubes and short exposures no untoward results had been seen.

The cases shown illustrated the following points:—

(1) Ulcerating forms of lupus rapidly heal up, leaving smooth scars.

(2) The treatment can be applied to mucous surfaces—*e.g.*, the interior of the nose, mouth, etc.

(3) In two of the three cases where the X rays had been applied daily for months lupus nodules were still visible embedded in the cicatrix. One patient, who had in other respects improved remarkably, showed nodules even after ten months' continuous treatment.

Digitized by Google

On this ground the X rays were considered to be inferior to the concentrated chemical rays of light.

Dr. Sequeira had found it advantageous to use the X rays to heal up ulcerated surfaces, and then when the pressure apparatus could be applied without pain, to treat the isolated nodules by the Finsen method.

DR. DORE also showed five cases of lupus vulgaris treated by himself and MR. MALCOLM MORRIS with the X rays and Finsen's method.

The cases were tabulated as follows:—

1. *Male. Aged* 21. *Duration of Treatment.*—About fourteen years.

Previous Treatment.—Cauterisation by chemical means, scraping, partial excision, thyroid extract, the original tuberculin, and tuberculin R.

Condition on Commencement of Treatment.—Extensive ulceration round mouth and both lips, much swelling of upper lip and disseminated nodules on cheeks and chin.

Duration of Finsen Treatment.—About five months daily, many intervals owing to severe reactions. Total number of exposures, 164. Also by X rays, seventeen exposures to different parts of the disease.

Result.—Healing of ulceration, with great reduction of swelling of the lips and disappearance of the greater part of the disease from the cheeks.

2. *Female. Aged* 32. *Duration of Disease.*—Sixteen years.

Previous Treatment.—Cauterisation, seven times; scraped in all thirty-four times, both varieties of tuberculin. Condition on commencement of treatment, large area of nodular lupus involving the whole of both lips and almost the whole of both cheeks, cartilaginous part of nose destroyed, lupus of lining membrane of nose.

Treatment.—X rays for six months, and 497 exposures to Finsen's treatment.

Result.—Complete healing of all parts with almost complete disappearance of the nodules.

3. *Female. Aged* 38. *Duration of Disease.*—Twenty-three years.

Previous Treatment.—Cauterisation, scraping, both kinds of tuberculin.

Condition on Commencement of Treatment.—The whole of the left cheek was involved, including the left ear, in a large patch of nodular lupus, which extended behind the ear on to the neck.

Duration of Treatment.—400 exposures to Finsen treatment from May, 1900, to June, 1901.

Result.—Disappearance of a great many of the nodules and subsidence of some of the swelling of the ear.

4. *Female. Duration of Disease.*—Since ten years old. Patient's age was not stated, but she appeared to be about thirty-five years old.

Previous Treatment.—Scraping and cauterisation.

Digitized by Google

Condition on Commencement of Treatment.—A large patch of scarred and crusted lupus on right cheek.

Duration of Treatment.—June, 1900, to July, 1901, by Finsen's method, in all 195 exposures.

Result.—Almost complete disappearance of all the disease.

5. *Female. Aged* 32. *Duration of illness.*—Fifteen years.

Previous Treatment.—Cauterisation by chemical means and heat; excision.

Condition on Commencement of Treatment.—Extensive lupus of face.

Duration of Treatment.—Three months; still under observation. In all, forty-five exposures by Finsen's method.

Result.—Disappearance of most of the nodules.

MR. STARTIN also showed a case of ulcerative tuberculosis of the skin of the face in a girl, aged 17. The patient had been treated with X rays only, and most of the ulceration had healed up under this form of treatment. Duration of treatment, thirty exposures of ten minutes each.

MR. BUCK showed two cases of tuberculosis of the skin, treated by the internal administration of urea in doses of from 30 to 60 grains three times a day.

P. T. *Age* 30. *Occupation.*—Chemist's man.

Family History.—Father died of phthisis.

Personal History.—Lupus commenced on right side of face nine years ago and spread under chin, healing as it went, except at junction of lobule of ear with face. Four years ago fresh focus on left side of nose, spreading to corresponding cheek. Four years ago tuberculous dactylitis in second phalanx of middle finger of right hand. Bone disease healed, but left tuberculous disease of skin of back of hand.

Condition on Commencement of Treatment.—Patches of lupus were typically nodular and somewhat raised. Largest on left side of nose, size of half-a-crown.

June 24th. Urea grs. 30 t. d. s. July 2nd. Great improvement, chiefly flattening of nodules. July 9th. Patches becoming smaller and fading away. Ulcers on back of hand beneath crusts are now healed. Patient still under treatment.

Note.—Urea by estimation, 7 per cent. Quantity of urine unknown.

H. W. *Age* 36. *Occupation.*—Newspaper seller.

Family History.—Unknown.

Personal History.—Tuberculosis left ankle at four years. Tuberculosis of both elbows at ten years. Amputation of left leg at ten years. Arthrectomy of both elbows at eleven years.

History of present Illness.—Lupus commenced in nostrils at twenty-four years, and rapidly spread on to the upper lips, cheeks and septum of nose. All treatment failed to check disease.

Digitized by Google

Condition at Commencement of Treatment.—The face showed a large ulcerating surface, extending from the centre of the hairy margin of the forehead to the outer edge of each eyebrow, and then down on to both cheeks, where the total width of the disease reached six and a half inches. The left lower lid, most of the upper lip, and the cartilaginous part of the nose had disappeared. Nasal bones, bony septum and turbinated bones had also been lost. Palate perforated. The edges of the ulcer showed typical lupus nodules, and the centre was covered with weak granulations.

Progress.—March 26th. Urea prescribed, with maltine. Ulcer dressed with ung. plumbi iodidi. First changes noticed after a fortnight's treatment. Changes comprised lessening of discharge, cleaning of ulcer, flattening of nodules, and tendency to heal.

April 25th. Improvement continued. Urea grs. 30 t. d. s. May 7th. Healing rapidly; edges of ulcer flat, except in one or two small areas. Urea grs. 50 t. d. s. May 27th. Ulcer nearly healed; several small nodules still present. Urea grs. 60 t. d. s. June 11th. Nodules on temple disappeared, those on lip disappearing.

Note.—The patient has taken maltine the whole time, but has not otherwise altered his habits of life. Urea estimation from time to time has shown 1·4 per cent. to 1·6 per cent. Urine passed about 40 ozs. Diet, chiefly potatoes.

Digitized by Google

ON THE VALUE OF THE RECTAL TEMPERATURE IN PULMONARY TUBERCULOSIS.

(With Six Charts.)

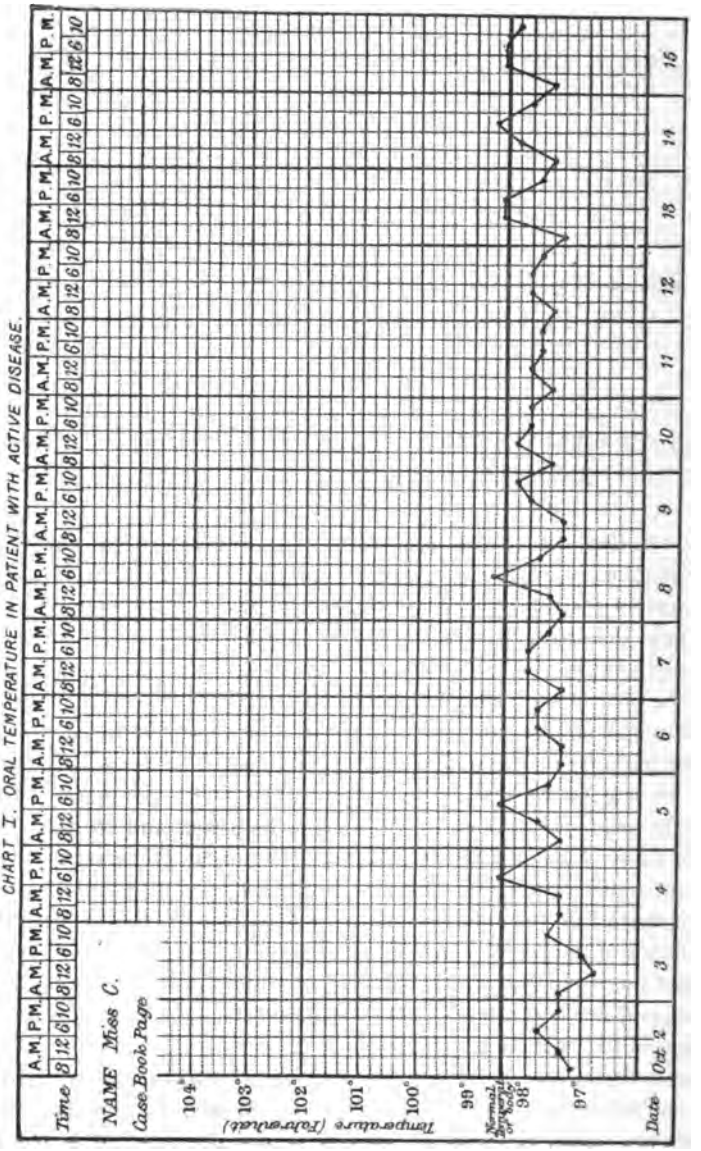
By J. C. BRAINE-HARTNELL, M.R.C.S. Eng., L.R.C.P. Lond.,
Resident Physician to the Cotswold Sanatorium.

No one can deny that a true knowledge of the amount of pyrexia in a given case of pulmonary tuberculosis is of the greatest importance, as it is, firstly, to a great extent the most reliable indication of the amount of active mischief going on in the lungs; and, secondly, one of the most important of data in giving a prognosis.

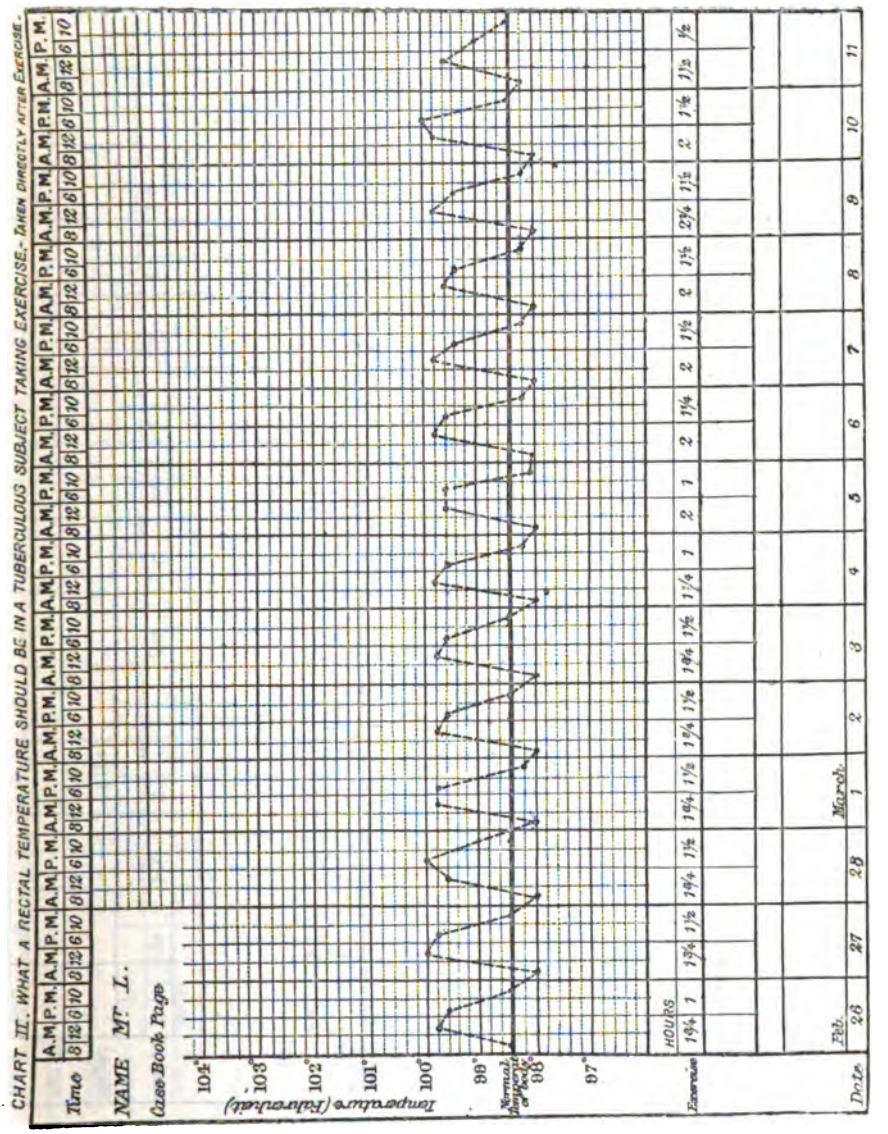
At the present time there is a great difference of opinion as to where the temperature should be taken—in the mouth, in the axilla, or in the rectum. The vast majority of medical men still hold to the time-honoured custom of taking an oral or an axillary temperature, but a few are more inclined to follow the plan carried out by Walther at Nordrach and to insist upon the superior value of the rectal temperature. The rectal temperature either gives important results and is therefore well worth the extra trouble and annoyance which it may cause patients, or it is a mere fad and unworthy of acceptance at the hands of the profession. The object of this paper is to endeavour to show that the rectal temperature is of grave importance, and that it is well-nigh imperative in cases of pulmonary tuberculosis, especially when patients are living in the open air all day long.

The æsthetic reason for not taking a rectal temperature, which is sometimes put forward, should not, we think, be allowed to bias our judgment when we are simply acting in the interest of our patients. It is surely a self-evident fact that if we are to control a patient's actions for the whole day and to regulate his walks entirely by the temperature chart, this temperature should be as free from error as possible. I believe that this is to be found in the rectum. It has been known for many years that the temperature in

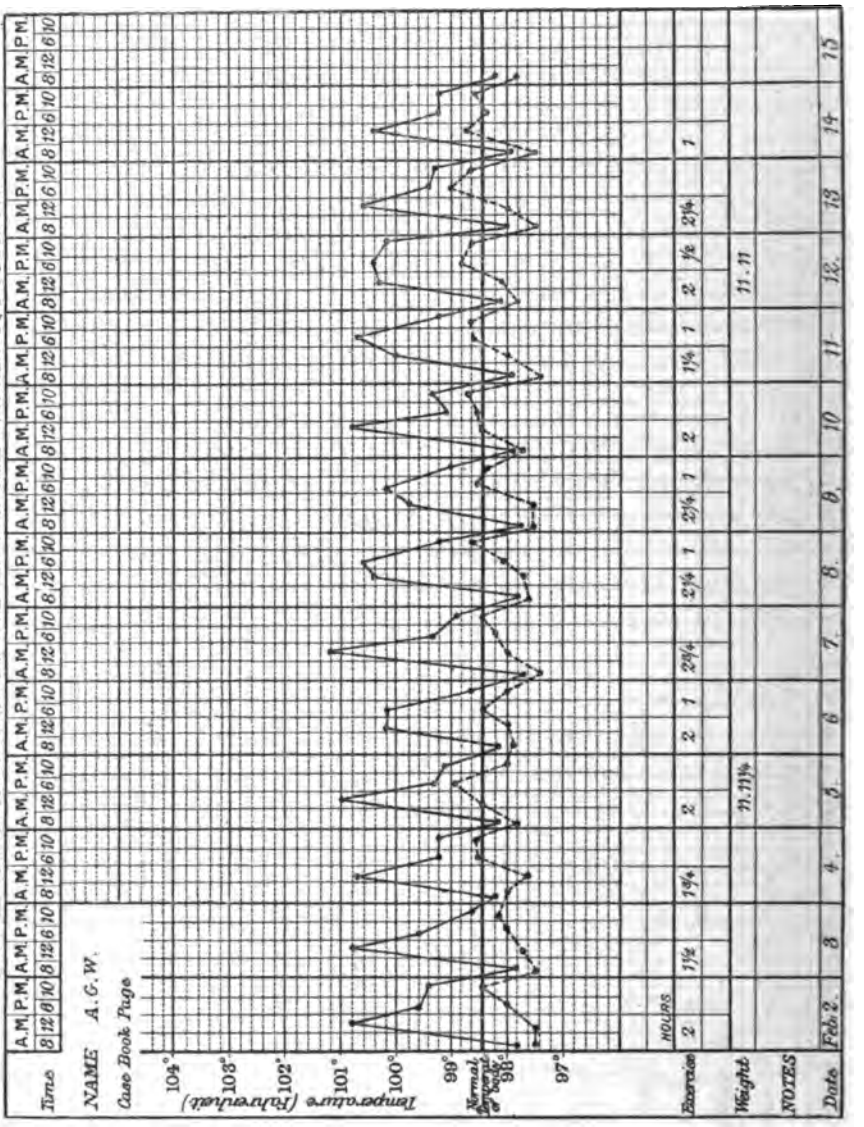
Digitized by Google



Digitized by Google

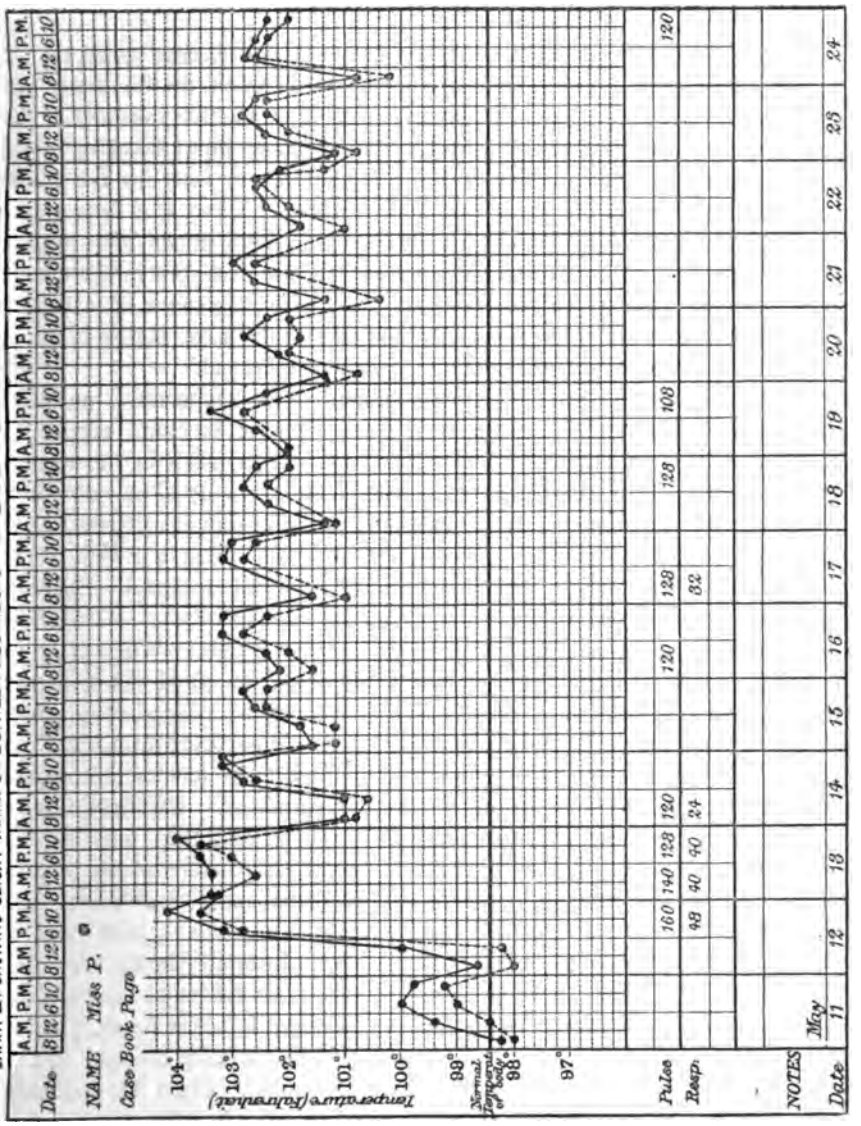


Digitized by Google



Digitized by Google

CHART IV. SHOWING SLIGHT VARIATION BETWEEN RECTAL & ORAL TEMPERATURES IN HIGHER RANGE.



Digitized by Google

pulmonary tuberculosis may be apyrexial if taken in the mouth or in the axilla. Without denying for one minute the occasional occurrence of active disease with apyrexia, I nevertheless believe that apyrexia is far less common than is generally held, and that many cases apparently apyrexial when tested in the mouth or axilla show, on the contrary, distinct pyrexia when the thermometer is placed in the rectum.

In three different patients, all of whom had active disease going on, the charts recording the oral temperature give no indication of the same. In the first, a patient with disease in both lungs and very active mischief at the left apex, physical examination revealed softening and rapid breaking down, but the chart denoted no rise in the temperature; in fact, for the whole fortnight the temperature never rose above normal. Chart I. is from a case with abundant physical signs of consolidation in both upper lobes with a cavity at the left apex. The temperature on only two occasions in the course of a fortnight rose to 99° F. In the third case, a patient of excitable temperament with active disease at the apex and at the base, during a whole fortnight there was no rise of temperature above the normal.

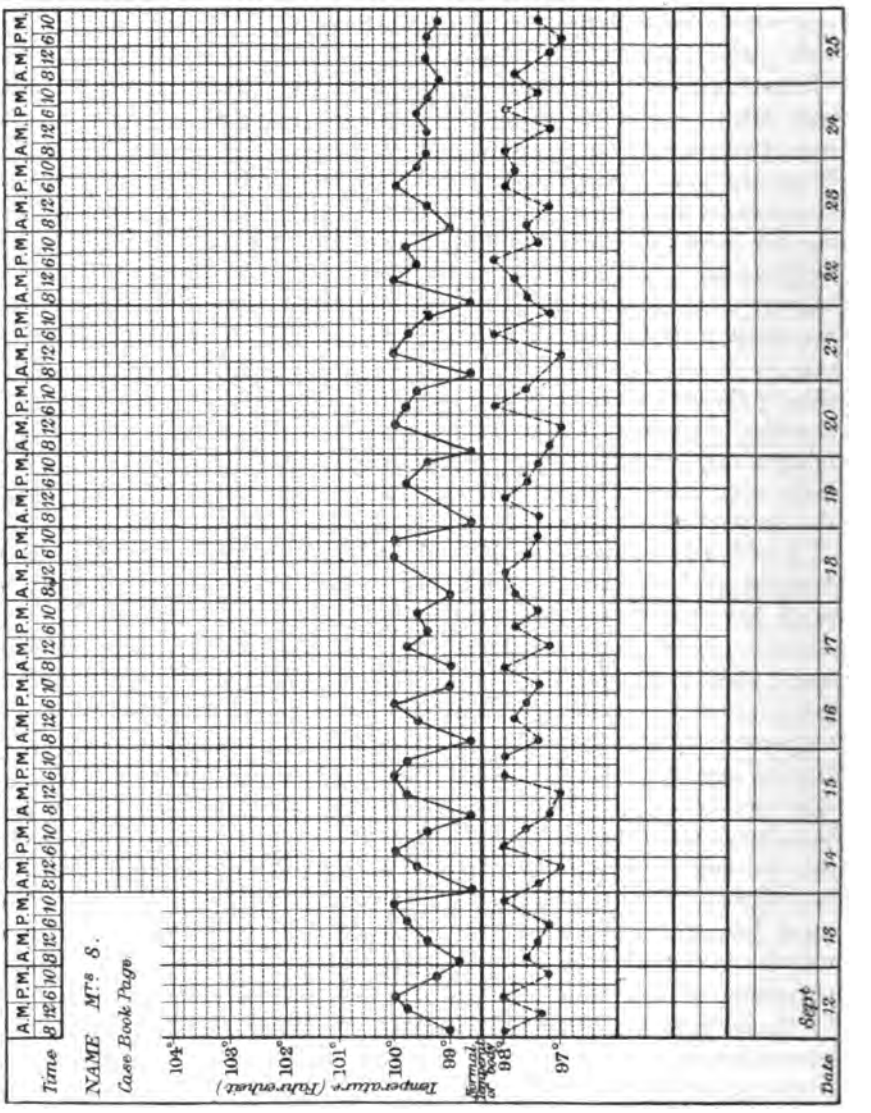
The question now arises, What is the normal rectal temperature, and what limits may be allowed in tuberculosis when exercise is being taken? I consider that the temperature of a healthy man when taken in the rectum should be normal or subnormal in the early morning—97·8° or 97·6° is a good morning temperature. After breakfast the temperature will rise to 99° or rather more, and exercise will have the effect of raising it a few points further. In the evening the temperature should again fall to normal. Exercise and food raise the temperature about one degree Fahrenheit. Wunderlich gives the average temperature for the twenty-four hours thus:—When taken in the axilla, 98·6°; taken in the mouth, from 98·78° to 98·96°; and in the rectum, from 99·14° to 99·5°. Guttmann again gives the average temperature for the twenty-four hours when taken in the axilla as being from 98·6° to 99·14°. As regards the limits allowed, I think that the temperature should be normal in the morning before walking is permitted. Certainly if any exercise is taken, it should be the very slightest, and then only on the level for, say, ten minutes at a time. A rectal temperature which is continually 99° in the morning, even though it keeps under 100° during the whole day, indicates activity and contra-indicates exercise, I think, and if rest be persevered with these temperatures almost invariably in the long run come down to normal. It is in these very cases that the rectal temperature is so important, as in very many the oral temperature is below normal, and if so guided, we are not likely to allow our patients to take exercise to the extent that we should if we followed the oral reading. Chart II. indicates what a rectal temperature should be in a patient who is doing well and is walking several miles a day. In this case there was disease in the left lung of fibro-caseous type and early laryngeal disease. The temperature after exercise was generally under 100°, and it dropped to under 99° if no exercise was taken. This is not an isolated case; in the cases of all patients who do

Digitized by Google

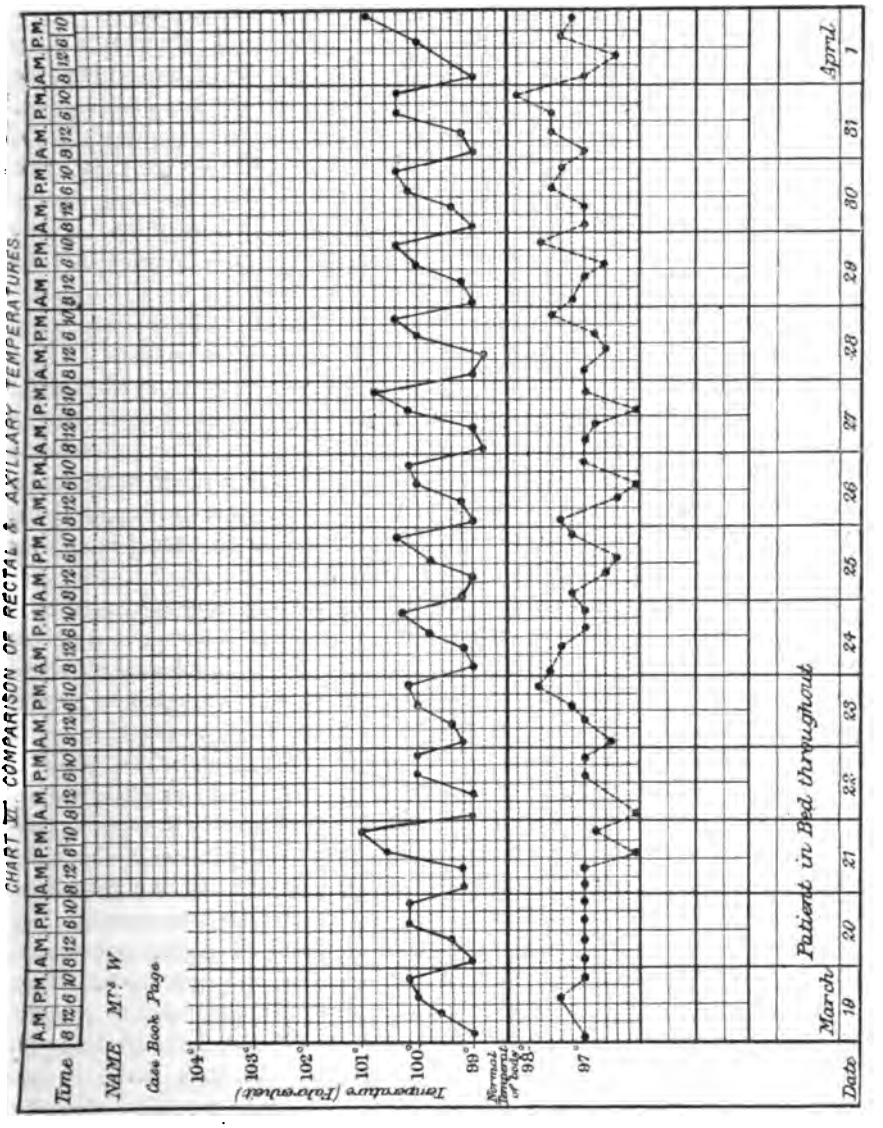
really well a chart similar to this may be expected to be found. Of course there are exceptions. The sanatorium treatment of tuberculosis demands individualisation in every particular, especially in feeding and in exercise, because if a patient is in other respects doing well there is no reason to restrict his walks if his temperature should rise to 101°. It may be idiosyncrasy or due to undue nervous excitement. Chart III. indicates this very well. The patient, who was exceedingly nervous, used to worry during the whole of his walk about his temperature lest it should be too high; in fact, he even went so far as to take his temperature during his walk if a suitable opportunity arose, with the result that it was almost always 101° or thereabouts. When sent out with a companion, so as to divert his thoughts from himself, the temperature was frequently almost a degree lower, clearly showing that the rise was simply due to nervousness. Again, I am acquainted with a typically healthy man whose rectal temperature is always over 100° after exercise, and between 101° and 102° after a ride on horseback. There are cases in which one must individualise. The height to which the temperature rises after exercise is not, in my opinion, of great importance provided that it drops directly the recumbent position is assumed. It is of the utmost importance that the temperature be taken directly after exercise, as a delay of fifteen minutes may cause the difference of one degree, and all observers who do not insist upon this will be led to believe that muscular exertion does not raise the temperature to any extent.

I wish now to show the value of the rectal temperature in tuberculosis. I cannot speak of other diseases, because I have had no experience of them, but I have heard Dr. Cayley say that he has seen no advantage from this method in enteric fever, and this is only what might be expected for reasons that I shall state presently. There is no doubt at all in my mind that in many cases of tuberculosis there is often no resemblance between the temperature taken in the mouth or in the axilla and that taken in the rectum. On the other hand, I acknowledge that in other cases there is little or no difference between the oral and rectal temperatures, though why this should be I cannot at present explain. This much I know, it is in the lower ranges that we find the greatest discrepancy, and the higher ranges the greatest resemblance. Thus in the case of a patient with early infiltration of the upper lobe of the left side, there was a great difference between the two records; in some records there is almost four degrees of difference, the rectal temperature rising after exertion and the oral falling as much as two degrees. On the development, however, of an acute caseous tuberculosis of the lower lobe on the left side the temperature rose in the rectum to 104·2° and in the mouth to 103·6°, and during the whole time that the temperature kept up there was very little difference between the oral and rectal temperatures, as shown in Chart IV.; but when with a subsidence of the acute symptoms the pyrexia diminished, the difference between the rectal and oral temperatures was as marked as ever. In the case of a female patient who was kept in bed for fourteen days consecutively no comparison between the two temperatures could be traced. The rectal record denoted active mischief, but it would be

Digitized by Google



Digitized by Google



Digitized by Google

difficult to demonstrate this from the oral record or even to diagnose tuberculosis unless one be led to do so by the subnormality of the temperature. In the case of a patient with infiltration at both apices, there was a typically good rectal record, the temperature being normal morning and evening, and with a rise on exertion to under 100°. The oral record was typical of nothing. In some cases there is great dissimilarity between the rectal and the oral records, the rectal in many instances rising, while the oral is falling to a corresponding degree. In others there is regularity of temperature, but always two degrees of difference between the rectal and the oral after exercise. Chart V. shows the dissimilarity between the oral and rectal temperatures in the case of a patient with considerable disease at both apices. I also think that a glance at Chart VI. will show how unreliable the axillary temperatures may be in some cases. The rectal record shows very strongly the activity of the disease, whilst the axillary record would mask any progress that the disease might make.

All the above records have been carefully taken for at least five minutes, the regulation time, and in most cases with new tested thermometers. I trust that by these charts I have been able to show the value of the rectal temperature. I think that mistakes are bound to be made in apportioning exercise unless the temperature is taken in this way. It is a very common experience to hear patients say that they have taken their temperature regularly at home for some weeks, and that they have no pyrexia, and yet when the rectal temperature is taken the thermometer in almost every case reveals a degree of pyrexia more or less marked.

Walther of Nordrach, as is well known, is very emphatic upon the importance of the rectal temperature, and so are all his followers. I fail to see how any man who has given this method a fair trial in tuberculosis could ever rely on any other; in fact, I am always afraid of being misled by an oral temperature—I mistrust it; but with a rectal temperature, on the other hand, I see exactly what degree of activity is going on, feel sure of my ground, and can be decided in the matter of exercise. With many patients it must mean all the difference between rest and exercise, progressive disease or convalescence. Exercise is one of the most important and yet one of the most difficult points to be decided; on that to a great extent hangs the great issue at stake. More patients probably come to grief through over-exercise than through anything else; it is to my mind the secret of the open-air treatment, the *crux* of the whole matter, when to allow exercise, and how much. If we made it a cardinal rule, and I most assuredly do so, that pyrexial patients must have no exercise, then surely we must know when we have to deal with pyrexia, and need not grope self-complacently in the dark with an oral temperature buoying ourselves and our patients up with the false idea that there is no fever, and therefore that all is going well, while all the time there is pyrexia, there is actual disease going on, and yet we know it not. It is so easy to take an oral temperature and to shut our eyes to the rectal, easy for the physician, and gratifying for the patients, because with a low temperature the treatment is not so irksome or so monotonous, and patients think that

Digitized by Google

they are doing well, which is half the battle, but they sorely resent being kept at rest for slight degrees of pyrexia as shown by the rectal record.

In conclusion, I think (1) that in many cases the oral and axillary temperature records are untrustworthy; (2) that in a few cases there is very little difference between the rectal record and the oral record; (3) that the greatest discrepancy is found in the lower range of the temperature, and conversely, the greatest similitude in the higher ranges; (4) that the rectal temperature gives in almost every case a truer idea of the amount of pyrexia; and (5) that if we wish to do ourselves, our patients, and our treatment justice we should insist upon taking the temperature in the rectum.

SOME OBSERVATIONS UPON DIETETICS IN PULMONARY TUBERCULOSIS.

By NOEL D. BARDSWELL, M.D., M.R.C.P. Edin. and Lond.,

Resident Medical Officer to the Sanatorium, Banchory, Scotland; late Clinical Assistant, Brompton Hospital for Consumption, London.

Dr. Kingston Fowler, in his address yesterday, referred to the work on dietetics and metabolism which I and my colleague, Mr. Chapman, are now engaged upon, and, although I had not intended to make any contribution on this subject to the Congress, at the request of the Secretaries of this Section I will endeavour to briefly tell you what we have done and what we are doing.

Over two years ago, when in charge of the sanatorium wards of the Sheffield Infirmary, two incidents led us to believe that we were overfeeding our patients, viz.:—

(1) That some patients who were taking very large diets and not gaining weight were excreting very large amounts of urea—over 900 grains daily. Such patients were mostly up to their normal weights and with quiescent disease;

(2) Owing to complaints of the expense of the sanatorium wards made by the hospital weekly board, we reduced the diets by nearly a third, and instead of disasters following, as we had feared, the results, as regards gain in weight and general improvement, continued to be excellent. On the strength of these two facts we fitted up the necessary apparatus and worked out the metabolism of one of the patients on a weighed diet of considerable dimensions. The results of this work, which were not published, strengthened us in our opinion then held—*viz.*, that the very large diets we were using were probably unnecessary and perhaps doing harm.

Last autumn, thanks to the courtesy of Dr. Fowler, who placed a ward in Brompton Hospital at our disposal, we, with the assistance of Dr. Goodbody, of University College, were able to carry out a fuller research upon the effects of ordinary and forced feeding upon the metabolism in tuberculous patients. The experimental work was carried out in Professor Harley's laboratory, so

Digitized by Google

that we had the benefit of his valuable advice all through the period of research.

To epitomise the results very briefly, we found that:—

(1) Patients when not closely supervised did not eat a sufficient amount of food, although plenty was given them, so that patients on the hospital routine improved quicker when placed under closer observation.

(2) The patients in the special sanatorium wards did excellently in every respect, judged both from a clinical and experimental standpoint. The diet of these wards is of practical interest, namely, three meals daily, made up of: meat, 7 oz.; bacon, 2 oz.; egg, 1; milk, 3 pints; butter, 3 oz.; bread, 7 oz.; sugar, 1 oz.; pudding, 5 oz.; vegetables, 6 oz.

(3) Patients originally on this diet, when given considerably larger quantities of food, such as 6 or 7 pints of milk, 12 or more ounces of meat, and in several cases with the addition of an ounce or two of somatose or lactose, did not give such satisfactory results. Weight was certainly gained more rapidly, but in several cases this gain was associated with deterioration of general health, and laboratory results indicating deranged metabolism. With regard to pushing proteid, we found that after a certain point much of the increased intake was wasted, in several instances as much as 50 per cent. of an increase being immediately excreted. Associated with this waste we found also a marked rise in the ratio of aromatic sulphates excreted, indicating increased intestinal putrefaction, and a steady fall in the per cent. absorption of nitrogen.

Coincidently with these experimental figures, indicating deranged metabolism, the patients' conditions, from a clinical standpoint, became less satisfactory. Loss of appetite and dyspeptic symptoms developed, and in one case vomiting occurred, symptoms all previously absent.

In contrast to the bad effects produced by giving a great excess of proteid, the inclusion of very large quantities of fat in the diet led to practically no diminution in the per cent. absorption of fat. This fact thus supports clinical experience as to the value of fat in the treatment of this disease.

For purposes of comparison, we then made similar experiments upon ourselves, and with somewhat similar results—*viz.*, rapid gain in weight at the expense of general health. The weight thus gained was rapidly lost on returning to ordinary feeding.

At the present time we are, at Banchory sanatorium, applying the results of this work to actual practice. Having carefully calculated out a diet for a new patient as probably suitable, the prescribed meals are weighed out, the leavings when necessary being also weighed, and the metabolism of these cases is periodically worked out in the laboratory. Careful clinical records as to progress of pulmonary lesions, etc., are also kept. From the results thus obtained, we hope soon to be able to speak more definitely upon this subject, but at the present time we believe that the very large diets often given in sanatoria are to a large extent unnecessary.

As an example of the practical value of studying dietetics in this disease,

Digitized by Google

I will conclude by giving a brief record of two cases at present under our care. One case is an example of dyspepsia and vomiting associated with underfeeding, and the other an example of similar symptoms associated with overfeeding.

(1) A. B., with extensive active disease in both lungs of some six months duration, very emaciated, and with pronounced dyspeptic symptoms; vomiting nearly every day. The diet for over two months has been approximately equivalent to: proteid, 70 grammes; fat, 160 grammes; and carbohydrate, 160 grammes, made up as follows:—

Breakfast.—Porridge with cream, 5 oz.; milk, ¾ pint.

At 11 A.M.—Milk, ½ pint, with egg, 1.

At 1 P.M.—Soup, 1 pint.

At 3 P.M.—Milk, 5 oz.; cream, 5 oz.; biscuits, 2.

At 7 P.M.—Benger's food, 1 pint.

She also took brandy, ʒi every three hours; maltine, ʒij; Fellowes' Syrup, ʒi; and creosote capsules, min. iv t.i.d., and in addition tonics and cough mixtures when she could manage to take them.

The results of this treatment, which was obviously directed to the amelioration of one set of symptoms rather than to the disease as a whole, were very unsatisfactory, the gastric symptoms amongst others becoming worse.

On admission the diet was increased 80 per cent. in proteid and 40 per cent. in carbohydrate, while the fat remained the same. Upon this diet the patient rapidly improved in every respect, the persistent vomiting being one of the first symptoms to disappear.

(2) The dyspepsia and vomiting associated with overfeeding. This type of case is not often seen in general practice, but appears to be fairly common in sanatoria. Such patients, in my experience, are usually up to or above their highest known weights, and with disease to a large extent arrested. They usually give a history of having done excellently on the high feeding for some months, but subsequently to have lost appetite and developed dyspepsia. The worst cases of this type suffer from great anorexia and nausea, usually intensified by the sight of food. They often vomit during meals, and not unfrequently bring up half a pint or so of greenish fluid before breakfast. This fluid contains no food.

The following are notes of such a case:—

C. D., with fairly extensive disease, largely arrested, apyrexial, and 4 lb. above his known weight, having gained over a stone as the result of thirteen weeks' sanatorium treatment. During the last six weeks, hitherto excellent appetite and digestion have given place to marked anorexia, dyspepsia, and general ill-health, and for the last month ¼ to ½ a pint of the greenish fluid already referred to has been vomited daily before breakfast. The diet for the past thirteen weeks has been approximately—proteid, 200; fat, 210: carbohydrates, 300; made up as follows:—

At 7 A.M.—Milk, ½ pint.

Breakfast.—Soup plate of porridge, with 1 oz. of cream; two courses of meat (about 4½ oz.); bread, 2 oz.; butter, 1 oz.; milk, ½ pint.

Digitized by Google

At 11.—Milk, ½ pint.

Dinner.—Soup; meat, 4½ oz.; boiled suet pudding, 5 oz.; vegetables, 5 oz.; milk, 1 pint; bread, 3 oz.; butter, 1 oz.

At 4 P.M.—A cup of tea, with bread and butter and milk, ½ pint.

Supper.—Two courses of meat (about 4½ oz.); vegetables and milk pudding (a large helping of about 5 oz.); bread, 3 oz.; butter, 1 oz.; milk, 1 pint.

On admission this patient's diet was reduced by some 30 per cent. proteid and 40 per cent. fat. The carbohydrate was unaltered. Walking exercise was considerably increased. Upon this treatment the dyspepsia and vomiting completely disappeared in ten days, appetite returned, and, what is more interesting, the patient gained a further 2 lb. in weight during the first fortnight, since when he has remained stationary at about 6 lb. above his highest known weight.

These are two extreme cases. A diet somewhere between the two just quoted would probably have given good results in either of the patients.

ÉTUDE ANATOMIQUE ET EXPERIMÉNTALE TOUCHANT L'INFLUENCE DE L'ALIMENTATION PAR LA VIANDE CRUE SUR L'ÉVOLUTION DE LA TUBERCULOSE EXPÉRIMENTALE.

PAR MM. LES PROFESSEURS CORNIL ET CHANTEMESSE.

LES bons effets de l'ingestion de viande crue utilisée comme méthode thérapeutique de la tuberculose ont été observés cliniquement chez l'homme par Füster (de Montpellier), et ont été surtout démontrés expérimentalement par les recherches de Richet et Héricourt sur les chiens; cependant les observations de Füster étaient tombées peu-à-peu dans le discrédit, et les faits signalés par Richet et Héricourt prêtaient le flanc à la critique. Leurs chiens mangeant à satiété soit de la viande cuite, soit de la viande crue, et la quantité de nourriture qu'ils absorbaient n'étant point connue, on pouvait penser que ces animaux mangeant plus volontiers de la viande crue, leur survie s'expliquait par les phénomènes heureux de la suralimentation générale, plutôt que par une action spécifique de l'albumine musculaire crue.

Pour élucider ce problème nous avons institué les expériences dans lesquelles les animaux nourris à la viande cuite ou à la viande crue recevaient en 24 heures le même poids d'aliment. Les chiens étaient partagés en lots de deux, chaque unité dans les lots ayant même apparence et même poids. L'un des animaux de chaque couple avait à satiété de la viande cuite; la quantité qu'il dévorait était mesurée chaque jour; et le second chien recevait, le lendemain, une quantité de viande crue égale à celle que le premier avait mangée cuite la veille. Dans chaque couple le régime du second animal était réglé par la régime du premier. La seule différence alimentaire résidait

Digitized by Google

donc dans la cuisson et dans la non-cuisson de la viande. Encore est-il à remarquer que pour obtenir un kilog. de viande cuite il faut environ 1250 grammes de viande pesée avant la cuisson ; il en résulte que les chiens nourris à la viande cuite absorbaient pour ce poids égal de nourriture une masse de viande plus considérable que celle fournie à leurs camarades soumis au régime de la viande crue.

Les inoculations de tuberculose ont été pratiquées dans la veine des animaux avec une culture très virulente, tuant le cobaye en six semaines, par inoculation sous-cutanée à la dose d'une anse de fils de platine. Tous les chiens ont été inoculés le 18 juin 1900, dans la veine de la patte, avec une dose égale d'une même culture.

Les résultats de ces expériences ont été décisifs. Les chiens nourris à satiété avec de la viande cuite ont succombé dans un espace de temps variant entre quatre semaines et trois mois ; à l'autopsie ils présentaient des lésions de tuberculose généralisée avec nodules caséeux plus ou moins volumineux.

Le foie était frappé de dégénérescence graisseuse avancée.

Le second animal de chaque couple, celui qui mangeait de la viande crue, était sacrifié aussitôt que mourait son compagnon nourri à la viande cuite. Dans tous les cas l'autopsie montrait la conservation d'un notable embonpoint, une dégénérescence graisseuse du foie modérée, et aussi de nombreuses productions de tuberculoses. Cependant les tubercules que l'on constatait étaient moins nombreux, moins volumineux et moins caséeux que ceux présentés par les chiens nourris à la viande cuite.

Nous avons gardé pendant un an, en continuant à ne le nourrir qu'avec de l'eau, un peu de soupe, et de la viande crue environ 750 grammes par jour, un de ces chiens dont le compagnon, qui mangeait de la viande cuite, avait succombé au bout de cinq mois. Pendant toute l'année, ce chien avait conservé sa force, son embonpoint et une santé en apparence parfaite.

Il a été tué par la chloroforme. Voici, avec les détails nécessaires, les résultats de son autopsie.

Nous avons trouvé des lésions de la plupart des organes du poumon, du foie, de la rate, et des reins.

Le poumon, libre d'adhérences pleurales, ne paraît pas congestionné ni hépatisé en aucun point. La surface de la plèvre viscérale laisse voir par transparence une zone mince, grise bien que semi-transparente, autour des vaisseaux sanguins. Des coupes pratiquées pour l'examen histologique après durcissement et montage dans la celloïdine, ont montré, autour des vaisseaux superficiels sous-pleuraux, et autour des bronches, des zones de tissu conjonctif infiltré d'un nombre plus ou moins considérable de petits leucocytes mononucléaires bien vivants dont le noyau était nettement coloré. Sur certaines coupes nous avons même trouvé des amas arrondis de ces éléments situés dans le tissu péribronchique ou périvasculaire, et représentant des granulations tuberculeuses fibro-leucocytiques, sans cellules géantes ni bacilles.

Le foie, en apparence normal, sans reliefs superficiels, sans induration, de

Digitized by Google

son tissu a été fixé dans l'alcool, durci et monté dans la celloïdine. Nous avons constaté sur les coupes beaucoup de petits amas de leucocytes mononucléaires le long des branches périlobulaires de la veine-porte représentant aussi des foyers de tuberculose ancienne fibreuse, sans bacilles, au moins dans nos préparations. Les ilots hépatiques sont sains.

La rate est de volume à peu près normal, plutôt un peu atrophiée. Elle est dure lorsqu'on l'examine sur leur coupe. On y reconnaît à l'œil nu des tractus blancs d'apparence fibreuse vus sur des sections longitudinales et transversales ; les sections transversales apparaissent comme de petits grains blanchâtres. Ces tractus très nombreux sont séparés par un tissu rouge, peu abondant, ne donnant pas de boue splenique à la pression ni au raclage, mais seulement un peu de sang.

L'examen histologique des coupes de la rate fait constater que les tractus précédents épais sont formés par du tissu fibreux dense et des faisceaux de fibres musculaires lisses, comprenant les artérioles spleniques dans leur épaisseur. Autour des artérioles, les corpuscules de Malpighi sont atrophiés ou invisibles. Le tissu splenique interposé à ces tractus est simplement formé d'une tissu reticulé rempli de leucocytes mononucléaires de petit volume et de lymphocytes. On ne trouve pas dans ce stroma de grandes lacunes ni de macrophages. C'est une splenite interstitielle sans vestige évident de granulations tuberculeuses.

Le péricarde, qui est si ordinairement atteint par la tuberculose chez le chien, n'y avait pas échappé, mas au lieu de granulations tuberculeuses, nous avons constaté une forme de péricardite chronique caractérisée par des productions végétantes, molles, fluctuantes sous l'eau, comme des houpes faciles à déplacer. Ces végétations étaient insérées sur le péricarde viscéral dans une étendue d'environ deux centimètres carrés ; elles ne s'étendaient pas au péricarde pariétal.

Après avoir fixé ces végétations par l'alcool et les avoir montées dans la celloïne nous en avons obtenu des coupes montrant leur implantation péricardique. Il n'y avait pas de granulations dans le tissu péricardique et sous-péricardique. Les végétations étaient constituées à leur centre par des fébrilles de tissu conjonctif servant de soutien à des vaisseaux ; elles étaient couvertes de couches de cellules endothéliales gonflées à protoplasma clair, à noyaux ovoïdes volumineux.

Le cœur était normal partout sauf dans l'artère pulmonaire, un peu au-dessus des valvules sigmoïdes. Là, en effet, on constatait une plaque saillante d'endartérite dans une étendue d'un centimètre carré. La saillie et l'épaisseur de cette plaque étaient assez grandes, un millimètre environ. Elle était, sur une coupe, un peu opaque, mais sans l'opacité jaune de l'athérome. Sa surface était lisse, sans trou d'ulcération. Les coupes perpendiculaires à la surface de l'artère a ce niveau ont montré que la lésion était due entièrement à la membrane interne du vaisseau. La structure consiste dans une infiltration très abondante de petites cellules rondes ou leucocytes mononuclées entre les fibrilles conjonctives de la membrane interne. Mais souvent ces éléments sont réunis en amas sphériques donnant l'apparence de

Digitized by Google

granulations tuberculeuses ; à ce niveau, la membrane externe présente des vaisseaux dilatés, la tunique moyenne est parcourue par des capillaires de nouvelle formation. La surface de la plaque est couverte d'un dépôt de fibrine et les couches superficielles de la plaque d'endartérite présentent une grande quantité de pigment sanguin de couleur orange, sous forme de grains plus ou moins gros.

Dans une autre partie de cette même plaque d'endartérite, la membrane de l'artère présentait des vaisseaux de nouvelle formation, quelques uns assez volumineux, de 0·05 mm. de diamètre par exemple, entourés de fibres conjonctives ; dans cette membrane interne, il y avait des ilots de petites cellules mononucléaires et un certain nombre de polynucléaires atrophiés reduits à des fragments de noyaux. Dans la couche interne de la tunique moyenne, on trouvait des ilots de granulations tuberculeuses avec des mononucléaires et des cellules de tissu conjonctif ; la partie externe de la tunique moyenne montrait ses faisceaux de fibres musculaires bien conservées entourées des cellules de tissu conjonctif.

Les préparations colorées en vue de la recherche des bacilles ne nous en ont pas fait découvrir.

Le rein est de tous les organes de ce chien le plus profondément altéré, non seulement par la présence de grosses granulations tuberculeuses, visibles à l'œil nu et contenant des bacilles, mais aussi par l'existence d'une néphrite chronique généralisée.

Les reins sont petits, rugueux et granuleux à leur surface, sous la capsule qui s'enlève avec une certaine difficulté. Ils présentent par places assez grandes des dépressions ou affaissements de la substance corticale dont la surface est chagrinée. Sur la section on reconnaît à l'examen microscopique des granulations semi-transparentes ou blanchâtres et opaques siègeant soit dans la substance corticale, soit dans la substance tuberculeuse. Ces granulations sont discrètes, de volume variable. Plusieurs dépassent le volume d'un grain de millet ; l'une d'elles est de la grosseur d'un grain de chènevis. Celle-ci est opaque à son centre. La muqueuse du bassinet est épaisse et très vascularisée, sans granulations ni érosions.

L'examen microscopique des granulations a montré que le tissu infiltré de leucocytes mononucléaires qui les constitue appartient au tissu conjonctif rénal, de telle sorte qu'à leur niveau, les tubes et glomerules sont écartés les uns des autres, et entourés par des bandes épaisses de tissu conjonctif rempli de leucocytes mononucléés vivants. Là, les glomérules sont aussi plus ou moins atteints, grossis par suite d'un dépôt de globules blancs entre la capsule et le bouquet glomérulaire ; ce dernier presentant aussi des lésions telles qu'épaississement de la paroi des capillaires, oblitération de ces derniers, etc. Les tubes compris dans la granulation ou situés a son voisinage présentent généralement une nécrose de leurs cellules ou diverses altérations de nephrite.

Pas de cellules géantes.

Les préparations colorées au ziehl et au bleu de méthylène pour la recherche des bacilles nous ont décelé, dans plusieurs coupes de granulations, quelques bacilles peu nombreux, deux à cinq par segment de granulation ;

Digitized by Google

ces bactéries étaient très bien colorées en rouge, comme le sont ces parasites vivants.

Dans tout le rein, les lésions de néphrite chronique sont des plus évidentes. Ainsi dans les parties superficielles affaisées, on voit des glomérules très rapprochés les uns des autres, presque tous avec une capsule propre très épaisse, fibreuse, se colorant très fortement en rouge par le réactif de Giesson. Le bouquet glomérulaire, toujours modifié dans le sens de la dégénérescence fibreuse, est parfois représenté seulement par un bouton fibreux. Dans d'autres parties du rein, on voit des ilots de tubes ayant conservé leurs dimensions normales entourées de bandes sclérosées dans lesquelles les tubes sont atrophiés.

Les pyramides de ces reins présentent aussi quelques granulations tuberculeuses interposées entre les tubes droits ou les englobant dans leur tissu néoplasique. Les vaisseaux sanguins de la base et de pourtour des pyramides, qu'il s'agisse d'artérioles ou de veines, sont tous atteints d'une sclérose très manifeste portant sur leurs tuniques externes et interne.

En résumé, d'après l'autopsie de ce chien, en apparence très bien portant, on peut dire que sa tuberculose était en excellente voie de guérison, mais qu'il avait conservé à la suite de la tuberculose du rein, une néphrite double chronique avec atrophie glomérulaire partielle, et avec des lésions scléreuses conjonctivo-vasculaires incurables qui auraient mis ses jours en danger au moindre accident pathologique survenu ultérieurement.

Nous avons tenté chez des animaux frugivores le traitement de la tuberculose expérimentale par l'ingestion de viande crue. Deux singes ont reçu dans les veines une culture de tuberculose très virulente. L'un a été laissé à son régime ordinaire ; il a succombé au bout de 23 jours à une tuberculose généralisée. Le second avait pris 15 jours avant l'inoculation, et avait continué depuis ce moment à ne manger que de la viande crue. Cette nourriture ne lui souriait pas, et il n'en prenait chaque jour qu'une petite quantité. Cependant, grâce à son régime alimentaire, il a survecu à l'inoculation pendant 49 jours.

Il résulte de ces expériences que l'utilité du régime de la viande crue utilisé pour le traitement de la tuberculose ne réside pas dans un phénomène de suralimentation pur et simple, mais qu'il repose sur une qualité particulière antituberculeuse de l'aliment.

The CHAIRMAN (Sir J. W. MOORE) : We are very much indebted to Professor Chantemesse for his short but excellent communication on the value of raw meat. We use it, and we all know its usefulness to tuberculosis ; and, if we want a further confirmation of this, we have only to look at how very rare tuberculosis is among the carnivora.

Digitized by Google

THE TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDREN BY RAW MEAT AND MUSCLE SERUM, ACCORDING TO THE METHOD OF MM. CH. RICHET AND HÉRICOURT.

By Dr. ALBERT JOSIAS, *Physician to the Hôpital Bretonneau at Paris*,

AND

Dr. JEAN CH. ROUX, *Ex-Intern of the Hospitals of Paris.*

We have carried out the treatment by raw meat and muscle serum, as described by MM. Richet and Héricourt, in a series of tuberculous children. Each of the patients received daily the expressed juice of 500 grammes of raw meat; cooked meat was banished from their diet, and an equal quantity of raw meat substituted. Treatment was strictly maintained for months, and, in some cases, for nearly a year.

Sixteen cases of pulmonary tuberculosis were investigated in this way, but of this number four have been left out of account, as they had only been under treatment for a few weeks.

The course of the remaining twelve has been followed with great care, observations being made almost daily, and the results are very accordant.

In only *one* case was the patient in the *first stage* of the disease (consolidation of the apex with some crepitation, but no breaking down). This patient is at present cured, her weight has risen by ten kilogrammes; in seven months she increased from twenty-three to thirty-four kilogrammes.

Four patients were in the *second stage* of pulmonary tuberculosis (more or less extensive infiltration, with softening, but no cavities). Two of these can be considered as cured after four months of treatment. In these patients all the signs showing a tuberculosis in process of evolution have disappeared. The condition of one is stationary from the point of view of pulmonary lesions; another patient improved, but left too soon.

Seven patients were in the *third stage* of pulmonary tuberculosis (infiltration with softening and cavities). The general condition of six of these was bad, and they were near the terminating period of the disease. In spite of treatment they grew worse, and death supervened in a period of time varying from one to three months after the raw-meat treatment was started.

One little girl only, in spite of advanced lesions and a cavity in the left lung, maintained a good general condition. After ten months of uninterrupted treatment she is evidently on the way to improvement.

Our conclusions can but confirm what we have said in a preceding paper. So long as we have to deal with a purely tubercular lesion, the patient markedly improves under the treatment; auscultation reveals an arrest of the tuberculous process, and the patient recovers. While saying, however, that

Digitized by Google

the cure can become permanent, it must be remembered that the future of the patients is the sole criterion of their complete recovery.

But when the tuberculous lesion is invaded by microbes which grow in the softening lungs and in the cavities, when the organism is radically affected by toxines circulating in the blood, treatment such as we have carried out has no longer any therapeutic effect in the large majority of cases.

RÉSULTATS DU TRAITEMENT DE LA TUBERCULOSE PULMONAIRE CHEZ LES ENFANTS PAR LE SUC MUSCULAIRE ET LA VIANDE CRUE.

PAR MM. LES DOCTEURS ALBERT JOSIAS (*Médecin de l'Hôpital Bretonneau à Paris*); JEAN CH. ROUX (*ancien Interne des Hôpitaux de Paris*).

NOUS poursuivons actuellement depuis plus d'une année des recherches sur le traitement de la tuberculose pulmonaire par la viande crue et le suc musculaire.

MM. Ch. Richet et Héricourt ont, en effet, établi la valeur de la viande crue ou du suc qu'on en exprime, pour arrêter l'évolution de la tuberculose sur le chien : notre but était d'appliquer à la clinique ces données expérimentales si séduisantes. Les essais ont été commencés à l'Hôpital Trousseau, et continués à l'Hôpital Bretonneau, à Paris, uniquement sur des enfants.

Tous les enfants tuberculeux qui entraient dans nos salles, quelle que fût la gravité de leur état, étaient mis aussitôt à ce traitement; tout autre médication était suspendue.

Nous avons déjà décrit, dans une précédente communication, le traitement, tel que nous l'appliquons à nos malades.*

Depuis le mois de juin 1900, jusqu'à la fin de février 1901, nos malades ont reçu, chaque jour, le suc extrait de 500 grammes de viande crue. Depuis le mois de février 1901, jusqu'à maintenant (juillet 1901) chacun des petits malades a reçu, en plus du suc musculaire, 100 à 150 grammes de viande crue par jour, dans du bouillon froid. La viande cuite était complètement supprimée de leur régime. En dehors de ces prescriptions l'enfant était soumis au régime ordinaire de l'hôpital.

La température des enfants était notée chaque jour, leur poids était pris chaque semaine.

Nous pouvons actuellement, en nous basant sur une expérience plus prolongée, préciser et confirmer les conclusions de notre communication du mois de février 1901.

---

* Albert Josias et Jean Ch. Roux: "Essais sur le traitement de la tuberculose pulmonaire chez les enfants par le sérum musculaire, suivant le procédé de MM. Ch. Richet et Héricourt." Société de Thérapeutique. Paris, février 1901.

Digitized by Google

I.

Nous n'avons eu la chance d'observer et de conserver à l'hôpital que trois malades au premier degré de la tuberculose pulmonaire, encore les deux derniers ne sont-ils avec nous que depuis fort peu de temps.

Voici résumées en leurs points principaux les observations de ces trois malades.

Notre première malade, H. Maria, est âgée de dix ans; elle est entrée dans le service le 6 septembre 1900; elle a toujours été assez délicate, et elle toussait souvent l'hiver. Lorsqu'on nous l'amène à l'hôpital, elle se plaint de céphalée, de douleurs abdominales, elle a une diarrhée très abondante. Les parents font remonter le début de ces accidents à une vingtaine de jours. L'enfant est très amaigrie, elle a une température irrégulière, qui oscille entre 38° et 39°; le ventre est excavé; la diarrhée intense, 10 à 12 selles par jour; il existe des douleurs dans les membres inférieurs; la rate est légèrement augmentée de volume et perceptible à la percussion. L'auscultation permet d'entendre des râles sibilants disséminés dans la poitrine; on note en plus au sommet droit, en avant et en arrière, de la submatité, une augmentation des vibrations thoraciques et quelques craquements.

Pendant tout le cours du mois de septembre, ces signes persistent sans se modifier sensiblement; la témperature baisse légèrement, mais la diarrhée continue et l'enfant s'affaiblit. Pendant le mois d'octobre, la diarrhée très abondante persiste, la fièvre, qui avait à peu près disparu, se rallume, et la température atteint jusqu'à 40°; l'enfant s'amaigrit et s'affaiblit de jour en jour. Le 7 novembre seulement, après l'échec des autres médications, on met l'enfant au suc de viande crue; l'effet est immédiat, la diarrhée s'arrête aussitôt pour ne plus reparaître, et l'état général commence à se relever. En six jours, la température revient à la normale et ne s'élève plus; le poids, qui était de 23 kg. 400 le 7 novembre, devient successivement de :—

23 kg. 800	... ...	le 9 novembre
24 kg. 100	... ...	le 12 ,,
24 kg. 600	... ...	le 15 ,,
25 kg. ...	... ...	le 19 ,,
25 kg. 500	... ...	le 23 ,,
26 kg. 200	... ...	le 28 ,,

L'augmentation de poids a été extraordinairement rapide; l'état général est excellent, l'examen du poumon ne permet pas de constater encore de modifications notables; il y a toujours quelques craquements et quelques râles sous-crépitants au sommet droit; la température est normale. Les jours suivants, le poids continue à s'élever régulièrement, et atteint 30 kg. 200 le 8 décembre. Vers cette époque, apparaît pourtant à la partie médiane du tibia, du côté gauche, une tuméfaction légère qui, au bout de quelques jours, devient légèrement fluctuante; il s'agissait probablement d'une ostéomyélite tuberculeuse à évolution lente.

M. Héricourt nous a, d'ailleurs, indiqué que, sur leurs animaux en

Digitized by Google

expérience, on pouvait voir parfois, alors que la tuberculose pulmonaire disparaissait, quelques localisations articulaires ou osseuses du bacille tuberculeux, localisations toujours bénignes, qui guérissent sans complication et qui indiquent la diminution de virulence du bacille.

En février 1901, l'enfant pesait 30 kg. 700, allait parfaitement bien ; la périostite était en voie de régression ; au sommet droit on trouvait encore de la submatité et la respiration rude, en avant et en arrière.

Depuis cette époque, son état s'est encore amélioré :—

le 1er avril, la malade pesait ... ... 33 kg. 100
le 1er mai, la malade pesait ... ... 34 kg. 400

poids qu'elle n'a pas dépassé, et auquel elle se maintient depuis deux mois. La petite malade a donc augmenté de 11 kilogrammes depuis le début de son traitement, en huit mois ; elle est devenue méconnaissable et jouit d'une santé florissante.

L'ostéite tuberculeuse, après avoir paru rétrocéder en février, s'est réveillée. La tuméfaction est devenue plus notable, la douleur plus vive, la fluctuation assez nette. Cet état a persisté jusque vers le commencement de juin. On n'a fait, d'ailleurs, aucun traitement spécial. Actuellement, il n'y a plus ni fluctuation, ni douleur, même pendant la marche ; le tibia gauche reste seulement un peu plus gros à sa partie moyenne.

Il ne reste rien au poumon, si ce n'est une expiration un peu rude et prolongée au sommet droit, en avant. Il y a longtemps que la malade ne tousse plus et ne présente aucun signe fonctionnel de tuberculose.

Chez cette malade, le traitement a donc produit les résultats les plus remarquables ; la disparition de la diarrhée, l'amélioration de l'état général, l'augmentation très considérable de poids indiquent l'arrêt dans l'envahissement de l'organisme par la tuberculose. Et la persistance de cette guérison, depuis plusieurs mois déjà, semble être la meilleure garantie pour l'avenir.

Nous avons pu soumettre au même traitement, il y a quelques semaines, un autre malade atteint également de tuberculose au début.

L'hom, Jul. ; âgé de 15 ans ; son père est mort tuberculeux. Un de ses frères est mort de méningite, une sœur de 23 ans est morte de tuberculose pulmonaire.

Depuis 6 mois, l'enfant se plaint d'un point de côté. Depuis trois semaines, il est malade, il tousse, il a une expectoration sanguinolente, il a souvent des frissons, surtout le soir.

L'examen des poumons n'indique que des lésions très peu avancées ; submatité, au sommet droit, en arrière, une respiration rude, légèrement soufflante. La toux est rare. L'enfant n'expectore que quelques crachats gris-verdâtre ; mais, dans ces crachats, l'examen bactériologique révèle de nombreux bacilles de Koch. Il n'y a pas de fièvre. La réaction à la tuberculine est positive avec 1 cc. d'une solution à 1·10,000ème.

Cet enfant est soumis au traitement suivant : 150 grammes de suc musculaire et 100 grammes de viande crue.

Digitized by Google

Sous l'influence de ce traitement, son état général s'améliore vite ; il semble en voie de guérison rapide :—

le 21 juin il pèse	...	...	...	38 kg. 400
le 30 juin il pèse	...	...	...	38 kg. 900
le 3 juillet il pèse	...	...	...	39 kg. 600
le 10 juillet il pèse	...	...	...	40 kg.

Broch, Louise ; 10 ans, entre dans le service le 19 février 1901. Depuis quelques semaines elle maigrit ; elle est enrouée et tousse un peu.

On trouve au sommet droit, en avant et en arrière, une submatité très nette ; et, à l'auscultation quelques râles sous-crépitants. Le côté gauche semble complètement indemne. La malade présente une légère réaction à une injection sous-cutanée de 1 cc. de la solution de tuberculine à 1·10,000ème. On la met aussitôt au traitement : 150 grammes de suc musculaire, et 100 grammes de viande crue. Le poids, qui était de 21 kg. 600 au début du traitement, est monté à 22 kg. 600 au bout de la première semaine, et à 23 kg. à la fin de la deuxième ; il atteignait 23 kg. 600 le 10 juillet. L'état général est excellent, la malade paraît en bonne voie.

En résumé, sur trois malades au premier degré, une petite malade, suivie longtemps, paraît guérie ; les deux autres, en traitement depuis quelques semaines, sont en bonne voie et marchent vers la guérison.

II.

Nous avons pu observer, pendant un temps suffisant, un nombre un peu plus considérable de malades au deuxième degré de la tuberculose pulmonaire, c'est-à-dire présentant une infiltration plus ou moins étendue du parenchyme pulmonaire, mais sans cavernes.

Les quatre malades, dont nous allons rapporter l'histoire, ont été suivis pendant des espaces de temps assez variables. Dans l'ensemble, les résultats sont très favorables, mais ils sont déjà inférieurs à ceux que l'on obtient dans la tuberculose au premier dégre.

Voici ces observations résumées :

Madeleine B., âgée de 8 ans, entre dans le service d'isolement de la rougeole, le 1er décembre 1900. Elle contracte une broncho-pneumonie, avec congestion pulmonaire intense et hémoptysie, qui évolue avec une température très élevée, oscillant entre 38°·5 et 39°·5, du 20 décembre au milieu de février. Il persiste à la base droite un foyer avec matité, râles sous-crépitants et un souffle. On trouve aussi quelques râles sous-crépitants à la base gauche.

On constate au sommet gauche de la matité, une respiration soufflante et quelques râles sous-crépitants, en avant et en arrière.

L'enfant est mis au traitement (suc musculaire 150 gr. et viande crue 100 gr.) le 10 février 1901.

La petite malade engraisse très rapidement.

Digitized by Google

Le 14 février le poids était de 19 kilogrammes. On ne trouvait plus que quelques râles sous-crépitants à la base droite et à la partie inférieure du poumon gauche. Au commencement de mars, la malade a un érythème polymorphe avec température élevée : 39° pendant 15 jours environ. Durant cette période le poids reste stationnaire, puis l'engraissement reprend ; et le 10 mars la petite malade pèse 20 kilogrammes. Le 30 mars, elle pèse 21 kg. 200 ; à ce moment, on ne trouve plus qu'une submatité légère au sommet gauche, en avant et en arrière ; la respiration est diminuée ; il y a quelques râles sous-crépitants en avant et en arrière.

Le 15 avril, le poids est de 22 kg. 500.

Le 15 mai, de 23 kg.

Nous notons à ce moment, pendant une quinzaine de jours, une entérite, avec fièvre et diarrhée, qui fait baisser légèrement son poids. Mais, cet incident passé, le poids se relève rapidement et il ne persiste plus qu'une diminution légère du son au sommet gauche. La malade ne tousse plus ; son état général est excellent.

Albertine F., âgée de six ans, entre dans le service le 28 janvier 1891. À l'âge de cinq ans, elle a eu la rougeole. Depuis cette époque, elle s'enrhume facilement ; cet hiver, depuis décembre, la toux a été plus fréquente, surtout la nuit ; en même temps l'enfant s'amaigrissait. Huit jours avant sa rentrée, la malade est prise de fièvre intense, revenant tous les soirs, et de sueurs nocturnes. Quatre jours auparavant, le 24 janvier, dans la nuit, l'enfant se plaint de nausées et rend, en toussant, un demi-verre de sang pur, très rouge, par le nez et par la bouche ; pendant la journée, elle rend encore quelques crachats striés de sang.

Au moment de son entrée à l'hôpital, l'enfant présente une température oscillant entre 38° et 39°. On trouve à l'examen du poumon : en avant, à gauche, de la submatité, de nombreux râles ronflants, des râles sous-crépitants après la toux ; à droite, une respiration rude et quelques râles ronflants.

En arrière : à gauche, au sommet, de la submatité, de gros râles muqueux et quelques râles sous-crépitants ; à la base, une matité de quatre travers de doigt, de nombreux râles muqueux et sous-crépitants après la toux, de la bronchophonie. Pas de pectoriloquie aphone.

L'enfant a perdu tout appétit.

On met aussitôt la petite malade au suc musculaire à la dose habituelle (500 grammes de viande crue par jour). Elle le supporte très bien. La température s'abaisse rapidement ; et, six jours après le début du traitement, la température atteint la normale et s'y maintient. Les signes de congestion du poumon droit et du sommet du poumon gauche disparaissent ; il ne persiste que la matité, à la base gauche en arrière.

La malade a présenté encore, par périodes, une fièvre légère, atteignant 38° le soir, jusqu'au mois d'avril. Mais les signes pulmonaires étaient très atténués : on ne trouvait plus qu'une submatité légère en haut et en avant du sommet gauche ; à ce niveau, la respiration était rude.

L'état général paraissait meilleur ; toutefois, le poids s'était à peine relevé.

Digitized by Google

Le poids, au début du traitement, était de 17 kg. 100. À la fin d'avril, il oscillait aux environs de 18 kilos. A ce moment la malade présente, pendant une quinzaine de jours, des râles de bronchite disséminés dans les deux poumons et une fièvre légère, atteignant parfois 38°, 38°·5 le soir. La toux était quinteuse.

Sous l'influence de cette poussée thermique, le poids diminue, il tombe à 17 kg. 200 et même à 16 kg. Mais la fièvre disparaît, la toux cesse, le poids se relève et atteint, le 20 mai, 17 kg. 400.

L'enfant était devenue un peu pâle; on ne constatait aucun signe inquiétant à l'auscultation des poumons; seulement un peu de rudesse au sommet. L'enfant est envoyée à la campagne.

La malade suivante est au traitement, depuis quelques semaines à peine, depuis trop peu de temps pour qu'il soit possible d'apprécier les résultats du traitement.

Berthe Lamp, 14 ans, enfant de souche tuberculeuse; sa mère est morte de la tuberculose il y a 8 mois; son frère est également atteint de la tuberculose; son père est éthylique. Elle tousse depuis deux mois, et a craché quelques filets de sang. Pas de fièvre.

A l'examen: Matité dans la fosse sus-épineuse gauche, submatité dans la fosse sous-épineuse; à ce niveau obscurité de la respiration et petits craquements à la toux. En avant, submatité sous la clavicule gauche et râles sous-crépitants de haut en bas. Rien à droite. La malade a une fièvre irrégulière, atteignant 38 à 39° le soir. La température reste à 37° le matin. Pas de bacille de Koch dans les crachats. La malade est soumise au traitement le 12 juin.

Pendant les quatre premières semaines de son traitement, on ne peut noter aucune modification de son état. La fièvre présente toujours les mêmes caractères; elle atteint 40° le soir; le poids, qui était, au début, le 12 juin, de 33 kg. 300, après s'être élevé à 34 kg. au bout de quelques jours de traitement, a baissé progressivement, et est actuellement de 32 kg. 200.

L'histoire de notre dernière malade est intéressante et instructive, bien qu'il soit difficile de la faire entrer dans la statisque, le traitement n'ayant pas été suivi d'une façon continuelle, pour des raisons indépendantes de notre volonté.

R., Jeanne, est âgée de dix ans et demi; elle est entrée dans le service le 27 mars 1900; elle tousse, depuis le mois d'octobre; elle a maigri, et a des sueurs nocturnes légères. Pendant le mois de janvier, elle a eu quatre hémoptysies; son appétit est conservé. On trouve, à l'examen, au poumon droit, en avant, de la submatité et des vibrations exagérées, une respiration soufflante, quelques craquements; en arrière, de la matité, dans les fosses sus- et sous-épineuses, des vibrations exagérées et, au même niveau, un souffle; la voix est très retentissante; il y a de la bronchophonie; il existe aussi des craquements humides et quelques frottements pleuraux. Dans le reste du poumon droit et dans le poumon gauche, rien à noter. Le ventre est souple, non douloureux, le foie de dimension normale.

Digitized by Google

À son entrée à l'hôpital, le 25 mars, la malade a une fièvre légère, mais qui diminue rapidement sous l'influence du repos; son poids, qui était de 26 kilogrammes, après s'être élevé à 26 kg. 500, retombe à 25 kilogrammes à la fin de mai.

Après avoir fait, pendant un mois, quelques injections de cacodylate de soude, qui ne modifient pas l'état général, et qui ne font pas augmenter le poids, on met l'enfant au traitement avec le suc de viande crue. Ce traitement est continué durant quatre mois; le suc musculaire ne fait pas disparaître toute élévation de température; par moments, la fièvre apparaît; la température atteint, le soir, 38 à 38°·5 pendant une dizaine de jours; on trouve deux poussées thermiques de ce genre, durant quatre mois de traitement. Le poids varie peu, il baisse plutôt légèrement, de 25 kilogrammes à 24 kg. 100, au moment de la sortie. L'enfant est cependant très améliorée. Elle ne tousse plus, elle a un aspect de santé parfaite et les parents la retirent malgré nous (25 octobre).

Nous revoyons la malade, le 7 février 1901. Chez ses parents, la petite malade n'a plus suivi son traitement avec la viande crue; la tuberculose a repris sa marche envahissante. L'enfant tousse dans la journée et vomit quelquefois, après ses quintes de toux. Son poids a diminué. Elle ne pèse plus que 22 kg. 900. Elle n'a pas de fièvre la nuit, ni de sueurs. Appétit diminué.

A l'examen: on ne constate pas d'adénopathie cervicale, ni sus-claviculaire. Il existe de la submatité dans la fosse sus-épineuse droite; partout ailleurs, sonorité normale. Bruit de pot félé sous la clavicule droite.

A l'auscultation, en arrière et à droite, nombreux râles sous-crépitants, pas de souffle, pas de gargouillement. En arrière et à gauche, respiration soufflante au sommet, pas de râles.

En avant et à droite, sous la clavicule, respiration soufflante, non caverneuse; nombreux râles sous-crépitants humides, sensation de gargouillement. En avant et à gauche, respiration normale, pas de râles.

Les lésions se sont étendues et aggravées pendant l'interruption du traitement. Les parents nous confient de nouveau leur enfant; on lui donne aussitôt 150 grammes de suc musculaire et 100 grammes de viande crue chaque jour.

On note tout d'abord une amélioration de l'état général; le poids se relève, de 22 kg. 900 à 23 kg. 900, en quelques semaines; et, la température, qui au moment de son entrée dans le service atteignait 38° le soir, revient à la normale.

L'état local du poumon ne se modifie pas. Après être resté quelque temps stationnaire, le poids recommence à baisser:—

le 7 mai	23 kg.
le 18 mai	22 kg. 600
le 25 mai	22 kg. 400
le 2 juin	22 kg.
le 10 juin	22 kg. 200

Digitized by Google

La température se relève ; la fièvre est assez vive, 38° le soir et 39 le matin.

L'état du poumon s'est aggravé ; au sommet droit, en avant, on perçoit un souffle caverneux ; la diarrhée s'installe pendant quelques jours.

L'état général est très mauvais. Très rapidement l'enfant s'affaiblit. Elle a des sueurs abondantes, de la cyanose au moindre effort. Les urines sont albumineuses. Elle est emportée par ses parents dans un état grave, désespéré.

Sur ces quatre malades, une seule, la première, peut être considérée comme absolument guérie ; les signes pulmonaires ont disparu, son poids a augmenté de 5 kilogrammes ; l'état général est parfait.

La deuxième malade est très améliorée, mais son poids n'a pas augmenté, ce qui nous oblige de faire quelques réserves pour l'avenir.

L'histoire de la quatrième malade, R——, Jeanne, précise l'importance de l'augmentation du poids. Durant son premier séjour à l'hôpital, elle avait été très améliorée, son état général était parfait, sa toux avait cessé, mais son poids n'avait pas augmenté. Or, trois mois après, elle nous revenait avec des lésions plus étendues ; et, malgré le traitement énergique qui fut institué, elle continua à perdre du poids et à s'affaiblir.

Quant à la dernière malade, Berthe Lamp, elle n'est avec nous que depuis quelques semaines ; mais, contrairement à ce que l'on voit dans la tuberculose au premier degré, le traitement ne paraît pas enrayer immédiatement la marche de la maladie : elle a encore de la fièvre, et elle maigrit.

Donc au deuxième degré de la tuberculose pulmonaire le traitement est parfois excellent ; mais, parfois aussi, il ne fait que retarder la mort. Les causes de cette différence d'action nous échappent complètement.

III.

Chez les tuberculeux au troisième degré, c'est-à-dire présentant une infiltration étendue avec des cavernes, les résultats sont beaucoup plus douteux. Dans la majorité des cas nous pensons que le traitement, tel que nous l'avons institué, n'a aucune action.

Dans un seul cas, chez une petite malade qui se trouve sur la limite entre le 2e et le 3e degrés, présentant une caverne qui s'est creusée pendant son séjour à l'hôpital, nous avons, au bout d'un an de traitement persévérant, obtenu une amélioration assez nette.

Voici d'ailleurs son histoire.

Maria P., âgée de dix ans, est entrée dans le service le 28 juin 1900 ; depuis un an, elle tousse, maigrit, a la fièvre ; elle aurait craché plusieurs fois du sang. Le jour de son entrée dans le service, elle a une hémoptysie.

On trouve dans le poumon gauche, dans le tiers supérieur, en avant et en

Digitized by Google

arrière, de la matité ; à ce niveau, la respiration est soufflante ; il existe des râles sous-crépitants dans toute la hauteur du poumon. La température varie entre 37°·5 et 38°.

Dès son entrée, le 28 juin, la malade reçoit, chaque jour, le suc extrait de 500 grammes de viande crue. Le 1er juillet, l'enfant pèse 21 kg. 700 ; le traitement est continué sans interruption jusqu'à aujourd'hui. Le 23 novembre, l'enfant pèse 23 kg. 100 : le 20 décembre, elle pèse 23 kilogrammes.

Pendant ces six mois de traitement, l'état de l'enfant est resté à peu près stationnaire ; l'état local n'a pas changé et l'examen du poumon révèle exactement les mêmes lésions qu'au début ; à cet égard, l'état de l'enfant ne s'est ni aggravé, ni amélioré ; l'état général n'a pas varié d'une façon appréciable.

La petite malade crachait souvent du sang avant d'entrer dans le service ; or, depuis six mois, cet accident ne s'est pas reproduit ; le léger état fébrile (38°) a disparu petit à petit, au bout de deux mois de traitement, et la malade a repris une température normale ; on ne note qu'une ou deux fois une poussée fébrile, durant deux ou trois jours, et pendant laquelle le thermomètre s'élève à 38°·5 ; cet état se maintient jusqu'au 6 janvier 1901. A cette date, la malade a une légère hémoptysie, peu abondante, qui se répète le lendemain, puis cesse définitivement. A la suite de cette hémoptysie, pendant une dizaine de jours, la température s'élève progressivement jusqu'à 39°·5 ; puis redescend à la normale ; l'enfant garde, d'ailleurs, son poids de 23 kilogrammes. À l'auscultation, on trouve une modification dans l'examen du poumon, l'état local s'est aggravé ; il existe au poumon gauche, en arrière, de haut en bas, surtout à la base, des râles sous-crépitants humides ; vers le tiers moyen, en dehors, on trouve un souffle cavitaire et des râles humides agglomérés (gargouillement).

Malgré l'existence de cette caverne, l'état local est resté assez bon. Dans les mois qui suivent, la température ne s'élève plus, la petite malade reste apyrétique et le poids oscille entre 22 et 23 kg.

À partir d'avril, les forces de l'organisme semblent se réveiller, le poids augmente lentement mais régulièrement jusqu'à aujourd'hui : il atteint successivement :

23 kg. 500	le 21 avril
23 kg. 700	le 12 mai
24 kg.	le 2 juin
24 kg. 200	le 30 juin

L'état général est meilleur. L'auscultation des poumons révèle des modifications nettes : les râles sous-crépitants disséminés dans le poumon gauche sont moins nombreux ; et, au niveau de la caverne les râles sont moins humides et moins nombreux. L'enfant est certainement en voie d'amélioration.

Chez les autres malades présentant, au moment de leur entrée dans le service, une infiltration étendue avec caverne et un très mauvais état général,

Digitized by Google

le traitement ne semble même pas avoir prolongé leur vie. Nous rapportons leurs observations résumées ci-dessous :—

	Lésions pulmonaires.	Survie depuis le début du traitement.
Prach, Marguerite .	Infiltration des deux poumons, surtout à gauche, avec cavernes.	26 jours.
Men, Marcel . . .	Infiltration des deux poumons. Caverne à gauche. Adénopathie considérable.	41 jours.
Delaf, Charlotte . .	Fièvre hectique ; sueurs abondantes. Mauvais état général. Cavernes des deux côtés.	12 jours.
A., Henriette . .	Infiltration étendue de toute la base gauche ; état cachectique.	30 jours.
L., Émile	Infiltration et ramollissement du sommet des deux poumons. Cachexie. Phlébite.	30 jours de traitement, puis emporté par ses parents dans un état désespéré.
Mans, Charles . .	Infiltration des deux poumons avec ramollissement.	21 jours de traitement, puis emporté par ses parents dans un état désespéré.
Humb, Yvonne . .	Infiltration du sommet droit avec cavernes.	13 jours de traitement, emportée par ses parents dans un état désespéré.

Tous ces malades, pendant le traitement, ont maigri, ou sont restés au même poids.

Chez ces malades, présentant des lésions étendues, avec infiltration, ramollissement et cavernes, le traitement ne paraît avoir aucune action.

IV.

Nous rapportons ici une observation d'une interprétation plus délicate, mais qui nous paraît offrir également un réel intérêt.

Louis Buhl, âgé de 3 ans et demi, fut amené dans notre service le 3 mai 1901, dans un état lamentable. C'était un enfant d'aspect malingre et chétif, rachitique, avec un thorax déformé et des nouures aux articulations. Il était très amaigri, avait un teint terreux et présentait un système pileux très développé, une sorte de duvet grisâtre qui, sur le front, s'étendait des cheveux aux sourcils et était particulièrement développé sur le dos et sur les cuisses.

D'après les parents l'enfant avait toujours toussé ; mais, depuis cinq mois, la toux était beaucoup plus fréquente.

A l'examen des poumons on constatait, au niveau de la base droite, en arrière, et dans toute la moitié inférieure du poumon, de la matité, et un souffle bronchotubaire, d'intensité moyenne, accompagné de râles sous-crépitants.

A la base gauche en arrière, submatité ; et, près de la colonne vertébrale,

Digitized by Google

quelques râles sous-crépitants peu nets. En avant des deux côtés, les poumons paraissaient normaux et intacts. La température, à peu près normale au moment de l'entrée, s'élève progressivement à 38°·5, 39°, 39°·5.

Etant donnés les antécédents du petit malade, et surtout son aspect cachectique, nous songeons naturellement à la tuberculose, et nous le soumettons au traitement ordinaire : 100 grammes de viande crue, 150 grammes de suc de viande.

Le malade supporte bien son traitement ; on note cependant une diarrhée, assez abondante, survenant par intermittence, mais qui peut aussi bien être mise sur le compte de l'infection.

Pendant les deux premières semaines, l'état général reste stationnaire, mais l'état pulmonaire s'améliore : le souffle s'atténue et la température tombe.

Quelques jours après, la température se relève, et l'on constate, au niveau de la base gauche, un souffle bronchotubaire, de la matité, et des râles sous-crépitants ; à la base droite, une atténuation de tous les signes antérieurement mentionnés.

Ultérieurement les signes pulmonaires du côté gauche s'effacent à leur tour, et, vers le 12 juin, la respiration est parfaitement normale dans toute l'étendue des deux poumons ; il ne reste aucune trace de l'inflammation si étendue.

A partir de ce moment, le poids, qui était resté à peu près stationnaire, se relève rapidement, et passe en quinze jours de 11 à 13 kilos.

Pendant l'évolution de toute cette maladie, l'enfant a pris régulièrement 150 grammes de suc musculaire et 100 grammes de viande crue.

Néanmoins, devant l'amélioration extraordinaire du petit malade un doute nous prend : nous nous demandons si nous n'avons pas assisté simplement à l'évolution d'une bronchopneumonie banale sur un petit cachectique.

Or, la tuberculine (injection de $\frac{1}{2}$ milligramme) donne une réaction des plus nettes. Il nous faut donc admettre qu'il s'agit bien ici d'un tuberculeux.

Mais, autour de lésions légères et impossibles à déceler aujourd'hui, s'était développée probablement une bronchopneumonie banale.

Il paraît peu vraisemblable, en effet, qu'une bronchopneumonie tuberculeuse ait rétrocédé aussi complètement et en si peu de temps.

V.

Somme toute, si nous faisons une statistique globale, comprenant tous les tuberculeux pulmonaires qui sont entrés dans notre service, même ceux qui étaient à la période ultime de leur maladie, nous restons en présence de seize tuberculeux soumis au traitement par le suc musculaire et par la viande crue. Actuellement, il y en a quatre qui peuvent être considérés comme guéris ; trois qui sont en voie d'amélioration ; un qui va mal ; huit sont morts.

Si nous faisons des groupes, suivant le degré d'évolution de leur tuberculose, nous aurons les classements suivants :—

Tuberculeux au 1er degré : 1 guéri ; 2 en voie d'amélioration.

Tuberculeux au 2e degré : 2 guéris ; 1 qui va mal ; 1 mort.

Tuberculeux au 3e degré de leur maladie : 1 amélioré ; 7 morts.

Digitized by Google

Cette statistique confirme ce que nous disions il y a déjà six mois. Tant qu'on a devant soi des lésions tenant seulement au bacille tuberculeux, le traitement par le suc musculaire et la viande crue à haute dose améliore notablement les malades; le poids augmente, l'état général se relève, les signes d'auscultation révèlent un arrêt dans l'extension de la tuberculose, le malade guérit cliniquement. Au temps de dire ce que cette guérison peut durer, car l'avenir des malades est le seul critérium de la guérison complète.

Mais lorsque la lésion tuberculeuse est envahie par tous les microbes qui végètent dans des poumons en voie de ramollissement et dans les cavernes, quand l'organisme est profondément lésé par les poisons qui circulent dans le sang, le traitement, tel que nous l'avons institué, améliore dans des cas très rares, mais le plus souvent n'a aucune action appréciable.

Dans ces cas avancés, c'est l'état général du malade qui nous paraît surtout régler le pronostic; si l'état général est bon malgré les lésions étendues, on peut encore espérer.

Cette notion concorde avec les hypothèses que nous pouvons faire sur le mode d'action du traitement par la viande crue. Il ne paraît pas que la viande crue jouisse d'une action directe sur les microbes de la tuberculose, ou sur leurs poisons; le meilleur argument que nous en puissions donner, c'est cette ostéite tuberculeuse qui a évolué lentement chez notre première malade en voie de guérison manifeste. Il semble plutôt que le suc musculaire et la viande crue agissent en augmentant la résistance de l'organisme, en facilitant sa lutte contre la tuberculose; le malade est placé dans de meilleures conditions, et guérit par les processus habituels de guérison de la tuberculose. L'augmentation rapide du poids, dès le début du traitement, est le meilleur indice de ce réveil de la résistance organique et, par suite, nous paraît être le signe le plus favorable pour le pronostic.

VI.

Nous ajouterons que nous avons essayé le traitement dans quelques autres localisations tuberculeuses.

Cinq enfants atteints de méningite tuberculeuse, mis au traitement par la viande crue, dès leur entrée à l'hôpital, ont succombé aussi rapidement que ceux qui étaient abandonnés à leur sort.

Il n'y a rien d'étonnant à cela; l'un de nous a montré, dans un travail en collaboration avec M. Ch. Richet, que, dans la méningite tuberculeuse expérimentale sur le chien, l'animal étant mis à la viande crue le jour même de l'inoculation, on n'obtenait une guérison que dans un quart des cas au plus.*

Une enfant atteinte de péritonite tuberculeuse avec ascite est restée trois mois au traitement, sans modification apparente dans son état; pas de diminution du volume du ventre, pas d'augmentation de poids; l'état général était peut-être meilleur à la sortie.

---

* Société de Biologie, juin 1901. Ch. Richet et Jean Ch. Roux: "Méningites tuberculeuses expérimentales, et leur traitement par la zomothérapie."

Digitized by Google

Une jeune fille avec péritonite tuberculeuse en plaques et fièvre, est au traitement depuis six semaines. La fièvre a baissé régulièrement; mais le poids diminue et l'état général est mauvais.

Enfin, un enfant de trois ans et demi avec tuberculose mésentérique, a été très amélioré; son poids a augmenté, de 11 kg. à 14 kg. 600 en l'espace de quatre semaines. Il s'agissait incontestablement de tuberculose mésentérique, puisque l'enfant a réagi nettement à une injection de 1 milligramme de tuberculine.

MEAT ALBUMIN DIETARY IN THE TREATMENT OF TUBERCULOSIS.

By F. M. FORBES ROSS, M.D. Edin., D.P.H., R.C.P.S. Lond.

Success in the treatment of tuberculous conditions, whatever may be the method for the time being which is most generally approved by competent authorities, must necessarily depend in great part on the problem of diet in this disease.

Particularly is this so with cases of pulmonary tuberculosis, in which defective alimentation is the most troublesome factor of the disease to be dealt with.

Defective alimentation may be understood, for all practical purposes, to be governed on the one hand by the digestive and assimilative powers of the sufferers; on the other hand, to be dependent on the easy digestibility of the food substances exhibited, that is, calculating the amount and quality of nutriment derived from the particular food by comparison with the amount of digestive effort expended, and its ultimate nutritive value and power of producing stable nutrition when finally assimilated.

The problem presented by the digestive and assimilative powers of consumptive patients is perhaps one of the most complex and difficult which the physician is called upon to face in the daily routine of practical medicine.

It is just those very articles of every-day dietary, of tried and proved food-value, which, owing to the peculiar circumstances of the consumptive, are most difficult and trying for his deranged digestive system to deal with, even in only a partially complete manner.

The ingestion of ordinary food, such as meat, vegetables, fats, etc., and the effort to extract from them their constituent proteids, carbohydrates, etc., in such a manner as to derive benefit therefrom, is often beyond the strength of the consumptive's naturally defective powers, and more frequently causes a tax upon his or her energies, which the small amount of nutriment derived is unable to compensate.

Almost any rational line of treatment adopted will prove satisfactory and equal to the demands made upon it, provided the digestion of the sufferer be good and remains so.

When digestive powers begin to give out, which will almost inevitably happen in the vast majority of cases, then the fight to make any progress which may be called favourable begins in grim earnest.

Digitized by Google

The above amply accounts for the successes, failures, and general inequalities of results obtained under any of the well-known methods of treating phthisis.

Nothing more exhausting and distressing can be well conceived than a continuous effort to derive nutriment from food, unsuitable in quality and quantity, under circumstances of deranged functions and irritative and morbid conditions of the digestive apparatus.

Energy is uselessly expended in a stupendous endeavour to extract an amount of benefit miserably incommensurate with the effort, and with a result which would only count as a minimum under more favourable conditions.

Comprised in the term "digestive power" is the ability of the nervous mechanism of the pulmonary-gastric chain to resist the mutually reciprocal stimuli which the various processes in the one set of organs excite in the other set, and *vice versâ*.

Marked additional gastric effort, especially accompanied by diminished digestive power as the result of functional or irritative morbid conditions, made more profoundly disturbing by these very conditions, tend to call into action the pulmonary reflexes, *i.e.*, cough; this last, if in itself sufficiently aggravated, reacts on the gastric reflex and leads to the manifestation of vomiting, and thus we here have set up a vicious cycle, ending in inability to retain food. As the ultimate result, the general nutrition of the patient rapidly fails, his or her powers of resistance to the disease are gravely diminished, and the disease makes swift and devastating progress towards an unfavourable termination.

As proof of the above contention that the easy excitability is due to loss of power, and that the deranged and irritable reflexes require strengthening, the benefit to be derived from small doses of a nerve stimulant such as strychnine, over and above a sedative such as opium, is well known and appreciated. The assimilative powers can be understood to embrace not only the ability to take up and absorb nutriment, but to fix it in the organism, and having done so, to be able to retain and keep it in that condition of potential stability and fixation.

The stability of the assimilated matter taken up into the organism depends much on the inherent kind and quality of the food exhibited.

A good instance of this is shown by the contrast in the behaviour of milk or casein albumin, and meat albumin or myosin albumin* as articles of diet in consumptive cases. Milk or casein albumin, where the gastronomic peculiarities of casein albumin can be borne, is taken up with very moderate rapidity, and benefit is obvious only after comparatively large quantities have been consumed. On the other hand, the beneficial results of the assimilation of milk albumin are rapidly dissipated under unfavourable conditions.

Meat albumin, on the contrary, quickly and readily manifests its better qualities in the early observable improvement in the sufferer, and the benefit so obtained is not so easily undone as in the case of casein albumin.

Whatever the benefits manifested in the young by a milk diet, the circumstances do not altogether hold good for the adult, and especially an adult

* *Lancet*, June 22nd, 1901. Forbes Ross: "Possibilities of Myosin Albumin."

Digitized by Google

needing the utmost possible assistance from food specially adapted for the needs of adult life, which is the case with the consumptive. Muscularity is the feature of the adult, therefore the proteid of muscle-food is more nearly adapted to the exigencies of that condition of life than the peculiarly adapted food of the infant, in whom muscularity is not the predominant note of development.

The above can be easily proved by anyone taking the trouble to weigh tuberculous patients fed on either diet mainly.

Whether this is due to the fact that the casein albumin of milk is an excreted or excretory albumin, and that the albumin of muscle is a fixed and more highly elaborated and differentiated albumin, or that the marked deficiency of albuminoid iron in the casein albumin accounts for the superiority of the iron-carrying muscle albumin, has not yet been satisfactorily settled.

Any benefits accruing from the ingestion of milk albumin can be easily assisted, and their stability enhanced and safeguarded, by the admixture and addition thereto of the albumins of meat, with their many and various good qualities.

Further, the resistant and combative forces of the patient to the ravages of the disease seem to be more markedly increased and fortified under a diet of meat albumin, or mixed meat and milk albumin, than under milk alone.

Personal practical experience of the results of feeding consumptive patients tends to strongly confirm the experience of others.

Meat albumin, or myosin albumin, can be used in the raw state, as a raw-meat juice made from fresh meat and used immediately after preparation. It can be made either by mincing the meat and expressing it, or by the cold extraction process with saline solution.*

This last can either be used alone as it is, or it can be flavoured with port wine, spices, etc.

Commercial raw-meat juice preparations on the market possess no advantage over the home-made raw-meat extract, and have the additional disadvantage of containing only a small percentage of actual albumin, are most expensive, and must necessarily be highly charged with chemical preservatives of more or less harmful nature. All the above methods of administering raw-meat juice, home made and commercial, run the risk of conveying tubercle bacilli from the meat from which they are made; and the meat juices of foreign origin may also contain the spores of anthrax bacilli, if made from meat where the cattle are subject to that disease.

On the other hand, meat albumin can be also utilised in a coagulated or cooked condition, and apparently with just as great benefit, judging from practical results. From elaborate experiments made by myself, it can be easily and safely put up in tins and sterilised ready for use at very small expenditure.

Héricourt and Richet's† researches and experiments with raw meat on tuberculous dogs induce them to attach some importance to the rawness of

* *Brit. Med. Jour.*, Oct. 13th, 1900. Forbes Ross: "Diet in Heart Failure in the Aged." *Loc. cit.*, and "Intestinal Intoxication in Infants." Forbes Ross. (Rebman: London, 1897.)

† Héricourt et Richet. 'Bulletin de l'Académie de Médicine.' Paris: Nov. 28th, 1899.

Digitized by Google

the meat—its action supposedly being due to a somato-antitoxine existing normally in muscle plasma or serum; actually the reason may really be that more albuminoid nutriment was forthcoming for less digestive effort.

Perhaps the contention that muscle-serum possesses antitoxic influences has some foundation in fact, as persons of well-marked muscular development are known to be less liable to consumption than others not so well developed muscularly. Consumptive patients of good muscular physique do better than those who are not so fortunate.

Finally, in cases of profound tuberculosis, such as the tubercular infection of a large serous cavity, as the pleura or peritoneum, the patients, however well developed muscularly, seem to lose their muscular tissue at a rate out of all proportion to that occurring in other diseases. This may be that the muscular tissue is used up in a vain attempt at combating the tubercular poison.

Djvorack * and Webber † speak well of, and recommend, a meat diet strongly in the treatment of phthisis.

At the present time, in my wards in hospital, is a well-marked case of tubercle of the right apex in a man who began to go down-hill rapidly and to lose weight and become anæmic, with cough and loss of all appetite, under ordinary diet with much milk.

He was put upon the albumin made from two pounds of good steak daily, and now, after three weeks, is so much better that all active, subjective, and objective symptoms have ceased and he expresses himself as feeling quite well. His condition is rapidly approaching the stage known as "arrest." ‡

About five months ago I withdrew from the right pleural cavity of another man three and a half imperial pints of fluid containing tubercle bacilli (Ziel-Nielssen, Honsell). After paracentesis he was full-fed and the right chest painted with iodine, but this case became one of dry tubercular pleurisy, spoken of so unfavourably by Maguire.§ He rapidly ran down, emaciated, and became weak and anæmic. He was now put on the albumin from one pound of meat daily, was sent into the country, and returned after three months, with a gain in weight of two stones, ruddy, feeling well, and with what was more important—his right pleura apparently well, but thickened. This is undoubtedly another case of "arrest" (*loc. cit.*) under meat albumin.

Another case of malaria with a specific taint and beginning tubercle of lung, though doing extremely badly with a most generous diet and appropriate treatment, yet is now, under meat albumin, as he expresses it, "a different man."

Another striking case was one published in the *Lancet* by Dr. Fletcher

---

* Djvorack. "Bericht über den Kongress zur Bekämpfung der Tuberculose." Berlin. p. 619. 1899.

† Webber. "Meat in Pulmonary Tuberculosis." 'Zeitschrift für Tuberculose und Heilstättenwesen.' 1900.

‡ Williams, C. Theodore. "The Arrest of Pulmonary Tuberculosis." *B. M. J.*, July 6th, 1901.

§ Maguire: Harveian Lectures. "Prognosis and Treatment in Pulmonary Tuberculosis." *B. M. J.*, p. 1566, Dec. 1900.

Digitized by Google

Little and myself,* which did wonderfully well with meat albumin and open-air treatment combined. This particular case had done badly in South Africa, whither he had been sent for cure, but returned much worse with tubercular laryngitis; yet in two months had so far recovered as to become a case of "arrest."

The assimilative powers of a consumptive are in direct ratio to the relative efficiency of the vital functions of his organism, *e.g.*, adequate oxygenation of blood leads to increased power of tissue storage and metabolism with withdrawal of pabulum from the blood stream; this, in its turn, leads to renewed demand for nutrient matter to replace that withdrawn, which is met by increased digestive effort of healthier tone.

The success of the open-air or fresh-air treatment of consumption is the fact that, digestion being improved and appetite sharpened, the patient is able to partake of just those articles of food which tend to provide him with nourishment best suited to his needs in return for the increased power of dealing with them.

The above factors produced the survival of the "high altitude" treatment, "change of air," and in a less degree, owing to lack of adequate food, the "long sea voyage" on a sailing vessel.

If it be our duty to the consumptive bread-earner† to provide him with sanatoria for the open-air treatment of phthisis, it also behoves us to endeavour to furnish him with easily assimilated foods within the reach of his pocket. No matter what method of treatment be found most successful medicinally, the factor of diet will ever remain paramount and essential to that method.

Myosin albumin returns a maximum of nutrient for a minimum of effort, peptonises rapidly, and is promptly absorbed, throwing little or no stress and irritating over-action on the stomach. When combined with fresh milk or stimulant extractives, it helps to ensure a highly nutritious food, and leaves additional power and spare energy for such valuable adjuvants to the dietary as extract of malt, cod-liver oil, bone-marrow, hypophosphites, etc.

THE RELATION OF ALCOHOLISM TO TUBERCULOSIS.

By T. N. KELYNACK, M.D., M.R.C.P.,
Of the Royal Infirmary and Owens College, Manchester.

Alcoholism and tuberculosis stand foremost amongst the conditions hampering human progress and limiting man's happiness. Through them the evolution of the race has long been impeded, and unfortunately, in spite of numerous restraining and restricting efforts, their baneful influence is still accountable for a high degree of mortality and an immense amount of sickness and suffering.

* "Open-air Treatment of Phthisis." *The Lancet*, Dec. 1st, 1900, p. 1567.

† Burdon-Sanderson, Sir J. "Our Duty to the Consumptive Bread-Earner." *B. M. J.*, p. 4, July 6th, 1901.

Digitized by Google

The stay of such calamitous states has long afforded problems which have appealed to all sorts and conditions of men, and their solution is yet to a great extent unaccomplished and remains surrounded by difficulties.

With two such morbid influences barring human advance, it was but natural that eager minds, studying the one, should have been compelled to recognise the presence and action of the other. Hence questions as to the association and true relationship of alcoholism to tuberculosis are of old standing. But since the answers have lacked in uniformity, it is particularly desirable that at least some approximation to the truth should be arrived at.

The position has been well expressed by Dr. Payne,* who says:—

"With regard to the influence of alcohol on the production of tubercle, the utmost divergence and indeed contradictory opposition of opinion prevails. Huss found tubercular phthisis to be rare in drunkards; and that has been the general conclusion drawn from post-mortem observations. It has even been thought that drinking freely checks the progress of phthisis, but of this I can find little evidence. On the other hand, the more general impression is that alcoholism is a frequent cause of consumption." And Dr. Payne goes on to say, "On this disputed point we must appeal to the methodised experience of those who have special opportunities of observation."

The subject, however, is one full of peculiar and unavoidable difficulties; and not the least arises from the prejudices which, however repugnant to scientific research, have only too frequently warped the minds of those who attempted an investigation of the matter. Efforts to elucidate the question by an appeal limited to what has been termed "clinical statistics" are necessarily permeated with fallacy.

Purely experimental research, although in many ways valuable and suggestive, nevertheless, in the presence of modifying influences concerning which we have imperfect knowledge and but limited control, is apt to lead to unreliable conclusions.

Mortality returns, although rendering material for interesting, and it may be useful analysis, afford data too gross for anything but rough generalisations.

A study by pathological examination of a large number of cases would seem to afford the most hopeful method of investigation, and when combined with discriminating clinical observations, is likely to accomplish as near an approach to the truth as we are able to obtain under present circumstances. But before indicating the manner and results of such a method of inquiry, it will be well to refer briefly to the opinions which various authorities, based on exceptional experience or special inquiry, have formed on the relationship of alcoholism to tuberculosis, particularly in this country.

Three views are possible, and each has had its advocates:—

(1) That alcoholism is antagonistic to tuberculosis.
(2) That alcoholism bears no special relationship to tuberculosis.
(3) That alcoholism definitely predisposes to tuberculosis.

* Payne: 'Transactions of the Pathological Society of London,' 1889, Vol. XL., p. 332.

Digitized by Google

I.—Alcoholism Antagonistic to Tuberculosis.

Before the true nature of tuberculosis became apparent, the opinion was somewhat widely held that the subjects of alcoholic excess seldom suffered from consumption or other forms of tuberculosis, and some went so far as to claim that the alcoholic state was antagonistic to tuberculosis. This impression still exists amongst a large section of the public, and probably in some measure is a survival of the opinion expressed by many of the earlier writers on pulmonary tuberculosis. Indeed, in some medical quarters such a view is still not altogether discarded.

The idea that alcoholism was in some cases a preventive of tuberculosis may, perhaps, in part account for the advocacy of its persistent and even excessive use in phthisis. Certainly the use of alcohol in cases of pulmonary tuberculosis has long been a vexed question, and even yet there is much confusion as to purpose and great variation in practice. Walshe apparently favoured the moderate use of alcohol in phthisis.*

Bennett† also, writing some thirty years since, said: "Of late years, in America, alcohol, especially whiskey, has been much lauded as a remedy in consumption;" but added, "I have seen a certain number of cases in which it had been long taken, but I cannot say with benefit. The daily ingestion of large quantities of nerve-stimulating, carbon-producing spirit certainly does not come under my ideas of hygienic treatment."

Flint‡ used alcohol freely, that is from six ounces to a pint of spirit daily, and appears to have considered it beneficial, "but not in the sense of exerting a special influence upon the disease."

Charteris, writing in 1877 concerning the administration of whiskey to phthisical patients, says, "In private practice I order it to be taken *ad libitum*."

Dr. Hermann Weber,§ in 1885, was convinced of the great usefulness of alcohol in the treatment of phthisis.

Dr. Thomas Harris‖ some few years since tried the effects of whiskey, containing 53 per cent. of alcohol, during a period of nine months. Twenty-six cases were so treated, the doses commencing with two drachms of pure whiskey, gradually increased up to one and a half ounces of whiskey every four hours day and night. Dr. Ransome, who saw many of the cases, expresses the opinion that "at certain times of the day the dose of alcohol was unnecessarily large, for some of the patients at times were distinctly flushed, and slept heavily even in the daytime." Dr. Harris states: "No case left the hospital in a worse state than that in which he entered it. As regards the physical signs, very little difference could be detected between those at

* Walshe. "Practical Treatise on the Diseases of the Lungs." Fourth Edition. London, 1871, p. 496.

† Bennett. "On the Treatment of Pulmonary Consumption." Second Edition. London, 1871.

‡ "Phthisis," by Austin Flint. London, 1875.

§ Weber, Hermann. *Lancet*, 1885, I., p. 606.

‖ "The Treatment of Phthisis," by Dr. Arthur Ransome. London, 1896, p. 95.

Digitized by Google

the date of admission and the date of the discharge of the individual." Manifestly in no instance did "cure" occur.

Reference has frequently been made to Stokes' account of a gentleman who recovered from pulmonary tuberculosis after regularly drinking "seven tumblers of punch every night"; but it is sometimes forgotten that at the same time he was to a great extent living an outdoor life, duck-shooting every day that he could go out, and, according to his own account, "had to stand up to my hips in the Shannon for four and five hours of a winter's day, following the birds." *

It is interesting to note that Bell,† of New York, as far back as 1859, opposed the view generally current, for he then stated: "The opinion so largely prevailing as to the effects of the use of alcoholic liquors, *viz.*, that they have a marked influence in preventing the deposition of tubercle, is destitute of any solid foundation. On the contrary, their use appears rather to predispose to tubercular deposition. When tubercle already exists, alcohol has no obvious effect in modifying the usual course it takes. Neither does it mitigate, in any considerable degree, the morbid effects of tubercle upon the system in any stage of the disease."

The abolition of alcohol from many modern sanatoria seems to indicate that, as Dr. Ransome‡ well expresses it, "Owing to the terrible consequences of excess, and to the proclivity of mankind to its excessive employment, it (alcohol) can only be recommended in selected cases, with many cautions."

II.

Alcoholism Unrelated to Tuberculosis.

Many hold that alcoholism bears no special relationship to tuberculosis; that it neither prevents nor predisposes to infection. This view seems to have been generally held by the older writers on tuberculosis.

In going over the literature dealing with phthisis and published prior to the discovery of the tubercle bacillus, one cannot but be surprised at the very few references to alcoholism as a factor of etiological importance. Even when mentioned it is generally in such terms as indicate that it is not considered as particularly influential.

Dr. Henry Ancell,§ writing in 1852, expresses his opinion that "a careful consideration of all the facts brought forward by the best-informed pathologists . . . leads to the conclusion that tuberculosis and the formation of tubercle is not one of the effects of alcohol, either in the human frame or in the lower animals."

---

* Stokes, W. Clinical Lectures on Fever. *Medical Times and Gazette.* London, 1854. Vol. xxx., p. 487.

† *Medical Times and Gazette.* London, 1859. i., p. 587.

‡ "The Treatment of Phthisis." By Arthur Ransome, M.D. London, 1896. p. 94. *See also* "Sanatoria for Consumption." By F. R. Walters. London, 1899.

§ A Treatise on Tuberculosis. By Henry Ancell. London, 1852. p. 456.

Digitized by Google

A similar opinion was expressed by Dr. Richard P. Cotton* in 1858 in his Fothergillian Essay, where he says, "It is worthy of remark that the habitual drunkard, he who is always in his cups, is not very often the subject of phthisis: such, at least, is the result of my own observations."

Many still contend that alcoholism can only be considered as related to tuberculosis in so far as it leads to a lowering of general vitality, and places the individual under conditions particularly favourable to infection.

III.

Alcoholism as a Predisposer to Tuberculosis.

The view that alcoholism definitely predisposes to tuberculosis has of recent years received much support. The tendency of modern opinion is to recognise in alcohol an agent which renders the tissue specially prone to tuberculous infection. This is well exemplified by several recent expressions.

Dr. Hector Mackenzie† believes that "alcoholism must be regarded as a powerful predisposing cause of tuberculosis. It is almost invariable to find tubercle present in the lungs in patients dying in the course of alcoholic paralysis. Tubercle of the peritoneum or pleura frequently complicates cirrhosis of the liver. Alcohol in excess undermines the strongest constitution, and renders the body less resistant to diseases of all kinds. This is especially true when the individual leads a sedentary town life. Considerable amounts of alcohol may be taken without obvious deterioration of health by persons who lead an out-of-door life and eat heartily."

"Alcoholic subjects appear to be specially liable to acute miliary tuberculosis."

One of the most recent and definite pronouncements is that of Professor Thomas Oliver,‡ who says:—

"Nor is there any truth in the opinion held by the laity, that the use of alcohol protects the individual against tuberculous disease. I do not refer to such a restrictive employment of alcohol as a glass of beer or stout, or a little wine at dinner-time. That may be helpful to the individual. It is to the mistaken opinion that where there is a family predisposition to the disease, it is necessary to be somewhat more indulgent. In my experience of young men with a hereditary tendency to phthisis, alcohol has, by the late hours which it encourages, and the careless and irregular habits that it fosters, frequently precipitated the individual into active tuberculous disease, which abstinence and more careful living would have assuredly prevented. It indirectly favours the development of tuberculous disease. Pulmonary tuberculosis was found by Dickinson to be more frequent in drinkers than in

* Cotton, R. P. "On Consumption: its Nature, Symptoms, and Treatment." London, 1858.

† Text Book of Medicine. Edited by Dr. G. A. Gibson. Edinburgh and London, 1901. Vol. I., pp. 352, 371.

‡ Text Book of Medicine. Edited by Dr. G. A. Gibson. 1901. Vol. I., p. 603.

Digitized by Google

ordinary people, in the proportion of three to one. Rolleston, quoting H. Mackenzie, states that in sixty-seven cases of pulmonary tuberculosis occurring in drinkers, a family history of tubercle was only found in ten, whereas it is found in about 30 per cent. of the ordinary cases. The frequent association of pulmonary tuberculosis with alcoholic peripheral neuritis, and its presence, too, in nearly one-third of the cases of cirrhosis of the liver, are circumstances which show that under the debilitating influence of alcohol resistance is reduced, ordinary pulmonary catarrhs are not so quickly thrown off, and thus the bacillus of tubercle gains an easier entrance."

Some have held that there is a tendency for phthisis in alcoholics to assume the fibroid form, but this is not in accordance with the best modern experience.

Drs. Poore and Allchin,* in a recent article, speak of the association of tuberculous phthisis with alcoholic cirrhosis of the liver as "well established; the course of the disease being usually one of rapid caseation and excavation with broncho-pneumonia and rarely towards fibrosis."

It is of interest to note that many years since the late Sir Benjamin W. Richardson † recognised the special proclivity of alcoholics to phthisis, and even went so far as to describe a special form of alcoholic phthisis.

The close association of tuberculosis and alcoholism has now been widely recognised, not only by British physicians, but also particularly by American and French observers, who appear to recognise more fully than has hitherto been the case in this country, that there are very important sociological and economical as well as medical aspects of the question.

Osler, voicing American experience, is very definite :—

"It was formerly thought that alcohol was in some way antagonistic to tuberculous disease, but the observations of late years indicate clearly that the reverse is the case, and that chronic drinkers are much more liable to both acute and pulmonary tuberculosis. It is probably altogether a question of altered tissue-soil, the alcohol lowering the vitality and enabling the bacilli more readily to develop and grow."

M. Maurice Letulle § has recently drawn attention to the frequent occurrence of pulmonary tuberculosis in the alcoholic workmen of Paris.

He believes that chronic alcoholism prepares the way for the development of phthisis, that it aggravates the tubercular condition when established, increases the gravity of the prognosis, and enfeebles the therapeutic action of hygienic and dietetic measures. These considerations he would have brought before the notice of the public. A campaign against tuberculosis, he holds,

* Poore, G. V., and Allchin, W. H. "Diseases Determined by Poisons."—"A Manual of Medicine." Edited by W. H. Allchin. London, 1900. Vol. II., p. 52.

† Richardson, B. W. *Medical Press and Circular*, March 17 and 31, 1875, pp. 224, 265; also "Diseases of Modern Life." Fourth Edition. London, 1877.

‡ "The Principles and Practice of Medicine." By William Osler. Edinburgh and London, 1898, p. 382.

§ Letulle, M. "L'Alcoolisme dans ses rapports avec la tuberculose pulmonaire."—*La Lutte Antituberculeuse*, 31 mai 1901, p. 25.

Digitized by Google

should be associated with anti-alcoholic efforts; and to this end the establishment of popular "anti-alcoholic restaurants" is advocated.

Thorain* also insists on the importance of adopting measures for repressing the progress of alcoholism as among the important prophylactic procedures for the stay of tuberculosis. M. Albert Robin also shows that alcoholism is capable of creating a soil for tuberculosis. M. de Lavarenne† has also recently discussed the association of alcoholism and tuberculosis. And now we have Professor Brouardel's testimony that alcoholism is a potent factor in propagating tuberculosis.

The evidence of Ruata‡ respecting Italy is interesting :—

"Alcoholism in central and lower Italy is almost unknown, while it is tolerably widely spread in Piedmont, Lombardy and Venice. In Milan, the most industrial town in Italy, possessing a large number of manufactories, alcoholism is widely spread, and the mortality from tuberculosis resembles that of England."

Various suggestions have been made to explain the special liability of alcoholics to tuberculosis.

There can be no doubt but that the non-hygienic surroundings of many chronic alcoholics afford very important conditions suitable to tuberculous invasion.

The vitiated atmosphere of the public-house, the uncleanly habits of many of the frequenters, the overcrowding and promiscuous congregation of individuals in varying states of health and disease, furnish circumstances peculiarly suited to the spread of tuberculosis. But, most important of all, the filthy and dangerous condition of many bars and beer-houses from the reckless deposit of sputum on the floors, where, quickly becoming dry, it mixes with the dust which is continually being raised by the moving crowd, furnishes a fruitful source of infection. Moreover, it must be remembered that many of the habitual frequenters of these places are often in an advanced stage of phthisis.

Having had exceptional opportunities of studying large numbers of cases amongst workhouse and hospital patients, I am convinced that the public-house must be considered as one of the most serious obstacles to the speedy stamping out of tuberculosis in this country. It is very necessary that sanitary authorities should recognise this most important aspect of the tuberculosis question. It should also be remembered that statistics have clearly demonstrated the liability of brewers and publicans to phthisis.§ Dr. John Tatham's valuable returns on the mortality from tubercular phthisis in England and Wales during the last forty years strikingly illustrate the heavy death-rate from phthisis in inn and hotel servants.

---

* Thorain. *Thèse de Paris*, 1894. See *Rev. des Sci. Méd.*, 1894, XLIV., 262.

† De Lavarenne. *Annales d'hyg. publ. et de Méd. lég.* Paris, 1901. No. 3, pp. 193-220.

‡ Ruata. *Medical Annual.* Bristol, 1901.

§ The Hygiene, Diseases, and Mortality of Occupations. By J. T. Arlidge. London, 1892.

Digitized by Google

But in addition to the danger of sinking into habits of excessive alcoholic indulgence, workers in public-houses are also subjected to the prejudicial influence of long hours in ill-ventilated apartments, necessary association with uncleanly and, it may be, tuberculous individuals, noise, excitement, anxiety, and the depressing influences of an environment where evidence of human misery and degradation are only too apparent.

The sluggish, loafing or sedentary habits of the chronic alcoholic, and the unfortunate circumstance that practically all drinking in this country is carried on indoors, must not be lost sight of. The movement to establish outdoor cafés may well be supported on hygienic grounds.

But the chronic alcoholic is probably always the subject of more or less nutritional impairment. Frequently there is a very conspicuous lowering of vitality, and in not a few instances states of pulmonary congestion or even hæmorrhage exist, which undoubtedly furnish a suitable soil for the development of the tubercle bacillus.*

Perhaps sufficient attention has not been given to the occurrence of states of ante-intoxication which undoubtedly prevail in many alcoholics. It is also well to remember that the chronic alcoholic, as the result of his habits, is frequently driven to engage in particularly unhealthy occupations; and his reckless indulgence commonly leads him to rest content with an overcrowded, dirty, dark and ill-ventilated dwelling, induces him to grow accustomed to an insufficiency of suitable food, and subjects him to the devitalising influence of depressing emotions.

It is, however, of interest in this connection to note that as regards the association of tuberculosis and malignant disease, an analysis of Manchester cases† indicates that the subjects of cancer do not show any special tendency to contract tuberculosis, and that malignant disease has little or no influence in reviving latent tubercular processes.

But it is contended by some that, even besides a general impairment of vitality and pernicious environment, there is a special prejudicial influence arising from the action of alcohol and its associates. This is a contention peculiarly difficult of proof. The evidence at present available is perhaps not altogether conclusive. There are, however, certain experimental facts which go to show that alcohol has a marked influence in altering or determining an alteration of the cells of animals subjected to the action of certain pathogenic organisms.

Professor Sims Woodhead‡ has shown from observations on animals that alcohol predisposes them to specific infectious disease.

Professor Abbott, of Pennsylvania, and also Dr. Delearde, working in the Institut Pasteur at Lille, have recently proved that alcoholised animals are more readily infected by many organisms than non-alcoholised subjects, and

---

* *See* Communication by Delépine: *Transactions*, Sixth International Congress, Hygienic and Demographic, Vol. II., p. 226.

† Kelynack, T. N. "Tuberculosis and Malignant Disease."—*Medical Chronicle.* Manchester, 1897. Vol. VII., p. 177.

‡ Woodhead, Sims. "Alcohol in its Medical and Scientific Aspects."—*British Medical Journal.* London, July 13, 1901.

Digitized by Google

other investigators have also shown that alcohol produces a marked negative chemiotaxis.

It has also been found that animals brought into a state of chronic alcoholism are much less readily rendered experimentally immune to microbial infection. Attempts have also been made to show that alcohol, through its influence on nervous structures, renders the tissues, and especially the lungs, prone to invasion by tubercle.*

IV.

In order to compare the views and opinions above indicated with our Manchester experience, I have analysed the results of pathological observation of a large number of hospital cases.

A reference to pathological data probably affords the most reliable basis for the formation of sound views as to the relationship of alcoholism and tuberculosis.

Some time since I collected a number of fatal cases of peripheral neuritis occurring in chronic alcoholics.† Eight cases were subjected to pathological examination during a period of three years. They formed 1·6 per cent. of all the medical cases examined during that period. Pulmonary tuberculosis was met with in seven. This gives a percentage of over eighty-seven. All the cases were females. In only one subject were there distinct tubercular lesions elsewhere than in the lungs, and there the intestines were also affected. In one subject there was an old tubercular focus at the apex of the lung, but it seemed probable that the active phthisis arose from a fresh infection rather than from this latent patch. In five cases both lungs were more or less involved; in two the left was the only one showing any distinct tubercular lesion. In three of the cases the course had been rapid. In four it seemed to have lasted for several months. One subject, it was stated, had "spat blood" six months before her death. In one case one lung only was studded with small tubercles, the injection evidently having been recent. In two there was more or less extensive caseation. Four presented evidences of softening and cavitation, and in two of these there was some fibrosis.

Since analysing the above I have gone over my more recent pathological notes. Two further cases of alcoholic multiple neuritis have come to autopsy. In one, a male, aged thirty-five, the subject also of hepatic cirrhosis, both lungs were studded with recent tubercles. There was no softening, but caseous foci at the apices were apparently of older formation than the tubercles elsewhere. In the other case, a female, aged thirty-two, in whom there was also a fatty and cirrhotic liver, the lungs, although congested, œdematous, and presenting patches of broncho-pneumonia, showed no distinct evidences of tubercle.

---

* *See* "Pulmonary Consumption, Pneumonia, and Allied Diseases of the Lungs." By Thomas J. Mays. New York, 1891. p. 61.

† Kelynack, T. N. "On the Occurrence of Pulmonary Tuberculosis in the Subjects of Alcoholic Neuritis."—*The Medical Chronicle.* Manchester, 1895–6. Vol. IV., p. 180. *See also* "On Multiple Neuritis in Alcoholic Subjects."—*The Medical Magazine.* London, July, 1901, p. 396.

Digitized by Google

Thus out of ten fatal cases of alcoholic neuritis, nine females and one male, eight—or 80 per cent.—were the subjects of pulmonary tuberculosis.

In 1895 I expressed the belief that "it is desirable to treat cases of alcoholic paralysis in special hospitals, in country houses, or in healthy private houses, rather than in the almost unavoidably tubercle-contaminated wards of a general hospital," and furthur experience has but strengthened this opinion.*

As far as is possible, outdoor treatment should be provided for all alcoholics, and it is most desirable that retreats for inebriates should in many respects, and particularly as regards opportunities for outdoor life, closely resemble the sanatoria for tuberculous cases.

In connection with the recent remarkable outbreak of arsenical neuritis in Manchester and district,† and occurring in the most serious forms in alcoholics among the poorer classes, it is interesting to note that in a considerable number of the fatal cases active pulmonary tuberculosis was present, and in some undoubtedly much hastened the end.

Tuberculosis is also of frequent occurrence in the subjects of alcoholic cirrhosis of the liver.

In order to throw light on this matter, the records of 3053 medical cases examined in the Pathological Department of the Manchester Royal Infirmary have been analysed.‡

One hundred and twenty-one examples of common cirrhosis of the liver were collected, and all doubtful and complicated cases omitted. Cases associated with cardiac or such chronic affections as seemed to have led to secondary changes in the liver were also excluded. All examples of "biliary" cirrhosis and those in any way connected with syphilis were passed over. After omitting several doubtful cases, in twenty-eight tuberculosis—either active, latent, or obsolete—was present. Thus over 23 per cent. presented evidences of tuberculosis. Active phthisis was present in fourteen, twelve males and two females; and active peritoneal tuberculosis in twelve, nine males and three females. Of these, six males and one female had involvement of both lungs and peritoneum. In five males and one female the lungs were affected, and in two males and two females the peritoneum alone. No less than twelve, or nearly 10 per cent. of the hundred and twenty-one cases, appeared to die directly from tuberculosis. The average age of those in which active tuberculosis was limited to the peritoneum was nearly forty-seven years. The average age of those in which active tuberculosis was limited to the lungs was just over forty-four years.§

I have also been much struck with the frequency with which so-called tubercular peritonitis is met with clinically in the subjects of hepatic cirrhosis.

---

* *Medical Chronicle.* Manchester, December, 1895.

† "Arsenical Poisoning in Beer Drinkers." By T. N. Kelynack and W. Kirkby. London, 1901.

‡ Kelynack, T. N. "Common Cirrhosis of the Liver: An Analysis of 121 Cases."—*Birmingham Medical Review*, 1897.

§ Kelynack, T. N. "On the Occurrence of Tuberculosis in the Subjects of Common Hepatic Cirrhosis."—*The Medical Chronicle*, 1896–7. Vol. VI., p. 262.

Digitized by Google

In many cases it is manifestly of the nature of a terminal infection, and the post-mortem characters of many of our cases have shown such to be the case.

I have recently communicated with all the retreats and reformatories for inebriates mentioned in the Report of the Government Inspector, with a view to the obtaining of data as to the frequency of pulmonary tuberculosis in such class of alcoholics.

Unfortunately, however, scientific records are apparently not generally available in such institutions. Of course the persons admitted are, as a rule, carefully selected, and presumably submitted to medical examination, and it may doubtless be taken that those presenting evidences of tuberculosis would be excluded. It is therefore particularly desirable that all such institutions should make returns as to the number of cases developing phthisis during their retention.

It is also to be hoped that those life assurance societies who have so-called "temperance" sections will in future afford particulars respecting the prevalence of phthisis amongst the non-abstainers as compared with the abstainers, as such are likely to be of much interest and undoubted value in indicating the relative liability to tubercular infection of the two classes.

Without going into a consideration of points connected with the morbid anatomy of the question, it may be admitted that tubercular processes as met with in alcoholics do not present any special or exceptional features. As in other states, there is very considerable variety in the exact character of the lesions. In many cases there is a rapid extension of tubercle, and the destructive process in the lungs often runs a rapid course. The pathological characters of the lungs in many of the cases I have examined have much resembled those met with in cases of diabetics dying with phthisis.*

In many cases, however, the onset is insidious and the progress accompanied by comparatively insignificant clinical manifestations, and hence the importance of very carefully examining all alcoholics suspected of being tuberculous.

At present the material for a statistical expression of the frequency of the association of tuberculosis with alcoholism is lamentably meagre, but such evidence as is forthcoming appears to furnish strong ground for the belief that measures aiming at the extermination of tuberculosis should also include means for the suppression of alcoholism.

TUBERCULOSIS OF THE HEART.

By H. NEWTON HEINEMAN, M.D., *Bad Nauheim (formerly of New York)*.

Omitting the common form of tuberculosis of the pericardium, which extends from chronic tubercular pleurisy by contiguity, I purpose considering tuberculosis of the endocardium, of the myocardium, and of the pericardium.

* During the nine years 1891–99, I acted as pathologist to the Manchester Royal Infirmary. Of 19 diabetic cases examined, 57·89 per cent. presented evidences of pulmonary tuberculosis. *See* "On the Occurrence of Pulmonary Tuberculosis in Diabetic Subjects."—*Medical Press and Circular*. London, June 26, 1901.

Digitized by Google

Tuberculosis in General.

Tuberculosis of the heart has been considered as of the rarest possible occurrence until Weigert * fixed attention upon the fact that, according to his experience at all events, in the majority of cases of acute general miliary tuberculosis, discrete small tubercles would be found in different portions of the heart.

The heart should more commonly have been found the seat of tubercular ulceration since Weigert, in 1877, gave to the medical profession the famous clue by which general tuberculosis (general infection) became possible, and is probably caused, pointing out the fact that tubercular ulceration of the pulmonary vessels existed in some of these cases, and that in others tubercular ulceration of the thoracic duct (Ponfick's † discovery) was found, and was the probable avenue of general infection.

The present more perfect staining methods (which we owe in part to Weigert) will certainly furnish us with a greater number of cases of heart tuberculosis in the future.

The idea of infection of the pericardium from the mediastinal and the smaller or larger bronchial glands, themselves previously degenerated, cheesy, and tubercular, is the result of Zenker's ‡ researches. This infection is transmission by contiguity.

So far as the mode of the infection goes in all cases, there is no reason to suppose that it is different from that in which the tubercle bacilli do their work in other parts of the body. Thus Wechsberg § sums up his work in the following sentences :—

1. Tubercle bacilli destroy by their poisonous action the cellular and interstitial tissue round about them.

2. They injure the newly-formed cells only to the extent that the connective tissue and vascular formation is interfered with, and that the protoplasm of these tissues is only partially destroyed (giant cells, spindle cells).

3. Finally the newly-formed cells themselves are completely destroyed (cheesy degeneration).

Tuberculosis of Endocardium.

Tubercular endocarditis was met with by Corvisart, Wagner, Potain, ‖ Rindfleisch, ¶ Lancereaux, ** Letulle, †† more completely described by Perroud, ‡‡ and latterly more exactly by Kundrat, §§ Cornil, ‖‖ Heller, ¶¶ and Weigert. ***

---

* "Sitzung d. Med. Section, Schlesischer Gesellschaft," July 13, 1877.—*Virchow Arch.*, Bd., 77–88.

† "Muenchener Natur-Forscher-Versammlung," Sept. 17–23, 1877.

‡ *Virchow Archiv.*, 96, p. 489.

§ "Zur Lehre v. d. prim. Einwirkung d. T. Bacilli." Weigert Laboratorium. Frankfurt, 1901.

‖ Cited by Hérard, Cornil et Hanot : "Phtisie Pulmonaire."

¶ Rindfleisch : "T. d. Histol. Path.," p. 240. Ed. franç., 1873.

** Lancereaux, "Atlas d'Anat. Path.," pl. 22, fig. 7 et 7', texte, p. 248.

†† Letulle : "Bull. d. l. Soc. Anatom." 1874. p. 537.

‡‡ Perroud : "De l'Endocard. Aiguë dans la Granule." Lyon *Méd.*, T. XIX. 1875. p. 1216.

§§ Kundrat : *Wien. med. Woch.* 1883.

‖‖ Cornil : *l'Abeille Médic.*, No. 51. 1884.

¶¶ Heller : *Centrbl. f. Bact.* Jan. 1, 1887. Bd. 1, No. 10, p. 30.

*** Weigert : *Arch. f. path. Anat. and Phys.* Bd. 77.

Digitized by Google

Tubercular endocarditis has been almost exclusively observed in cases of acute general miliary tuberculosis. Usually it gives no physical signs during life, because the rapid progress of the disease gives no opportunity for their development. At the autopsy two forms are met with: first, that of granules or nodules; secondly, that of cheesy masses. The latter form is the less frequent.

Letulle met with the latter form once. Near the septum of the left ventricle he found two masses, hard and yellowish-green, one on the upper border of the left ventricular wall, the other on the summit of a papillary muscle of the mitral valve.

Perroud found the nodular form quite common in children who had died of acute miliary tuberculosis, and especially of tubercular meningitis. The mitral valve is the favourite and almost exclusive seat of this form of endocarditis. On the border of the auricular surface of the two valves, a few millimetres distant from the attachment of the tendinous chords, we find vegetations which are formed of minute nodules, and may be discrete or confluent. The tubercle bacilli are usually found in the superficial layers.

It must, however, be borne in mind that not all endocarditic nodules in tubercular cases are tubercular.

As to the manner of infection, the mode of transmission already referred to deserves a brief repetition here.

Although Mugge first described a case of tuberculosis of the pulmonary artery and vein in a case of acute general miliary tuberculosis, it is Weigert's work, already referred to, that cleared up the mystery of the path of infection. In twelve cases dead of acute general tuberculosis, he found ten cases of tuberculosis of the pulmonary vessels, and two cases of tuberculosis of the thoracic duct.

In the pulmonary veins the disease appears in the form of thrombi or polypoid vegetations. These vegetations occupy the entire thickness of the wall of the vessel and take origin in a caseous centre without. Weigert considers these vegetations the starting point of the infection of the blood in acute general miliary tuberculosis, and thus, as far as our theme is involved, furnish the starting point for infection of the heart when this is involved in such cases, and it usually is so involved.

Lion,* of Paris, in an able essay on "experimental endocarditis," experimentally proved the mode of infection, its presence and the character of nodules in tubercular endocarditis, demonstrating that care in staining methods often determines not only the question of their existence, but also of the tubercular character of the nodules, even when invisible to the naked eye. Careless staining, and even indifferent work, are certainly responsible for many negative results.

Tuberculosis of the Myocardium.

It is quite interesting to note the discrepancy of opinions prevailing with reference to the occurrence of tubercle of the myocardium. Kreysig, writing

* Dr. G. Lion: "Essai sur la nature des endocardites infectieuses." Paris, 1890. Steinheil.

Digitized by Google

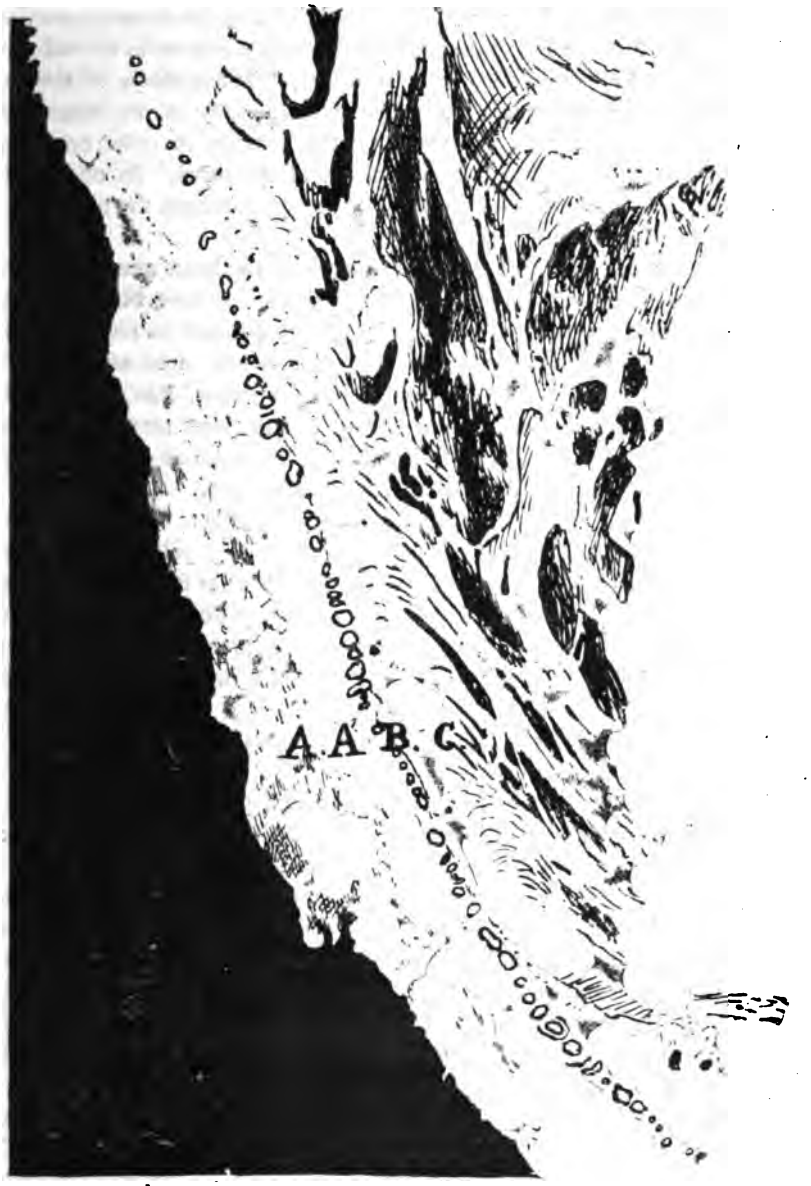


DIAGRAM OF PORTION OF SECTION OF HEART (ENLARGED).

Showing **A**, fibrinous layer; **A¹**, layer of tuberculous granulation tissue; **B**, layer of cheesy tuberculous nodules; and **C**, muscle wall.

Digitized by Google

in 1814 in Berlin, says,* "Tubercular tumours of the heart walls, while met with very rarely, are very probable."

Piorry † said (twenty-five years later), "Tubercles of the heart are very rare Laënnec found three or four cases in which tubercles existed in the substance of the heart." Still further he says (in 1840), "Tuberculosis of the heart succeeds a general tuberculosis of the body, especially of the lungs. The rarity of heart tubercles is explained by the scarcity of cells or cavities (hollow spaces) between the muscular fibres of the heart. In one case in which a tubercular abscess had opened into the pericardium the tubercles lay outside of this membrane."

Friedreich,‡ in 1867, said, "Tubercle occurs in the heart muscle in miliary form, in combination with miliary tuberculosis of other organs, often presenting such fine nodules that without the closest scrutiny one fails to recognise them, but appears under these circumstances and in this way more often than was hitherto generally believed (*v.* Recklinghausen, Virchow, Klob). The large tubercular or cheesy nodules are met with in the heart muscle but rarely, and, in fact, only in combination with chronic tuberculosis of other organs, especially of the lungs." (E. Wagner, Lüken, Waldeyer.)

Schrötter, in 1876, says in Ziemssen's Cyclopædia: "Tubercle of the heart occurs principally in the form of discrete miliary yellowish or greyish nodules in the connective tissue between the muscular fibres, aside from its occurrence as part of a general tuberculosis. Rarely do large cheesy nodules appear, and when present are a consequence of chronic tuberculosis usually penetrating from the vicinity (Waldeyer, Recklinghausen. Virch. Archiv. XVI. 1859)."

"Townsend § found (1832) a large tuberculous nodule starting from the left auricle and compressing the pulmonary vein."

Hartog, ‖ writing in 1901, says: "It seems a pity that Weigert has not received the recognition due him in clearing up this particular subject and the process of infection in acute miliary general tuberculosis."

A year after Weigert called attention to the way in which cardiac tuberculosis had been overlooked, Sanger, in 1878, still insisted upon his own views, but a year later (1879) admitted Weigert to be correct, when the latter said "acute general miliary tuberculosis is always accompanied by cardiac tuberculosis."

Form in Myocardium.—The forms in which tubercles appear in the myocardium are most commonly the fine transparent miliary nodules, at times greyish or yellowish, discrete or aggregated, as found in acute general miliary tuberculosis; again in the larger yellowish nodules, from pea to walnut size; rarely in large cheesy masses; least commonly of all as diffuse

* Kreysig, D. F. L.: "Krankheiten des Herzens." Berlin, 1816. 1 Theil, p. 360.

† Piorry, P. A.: "Über Krankheiten des Herzens und Gefässe." Translation of G. Krupp. Leipzig, 1844. p. 231, § 599.

‡ Friedreich, Prof. N.: "Handb. d. spec. Path. und Therapie." Erlangen, 1867. "Krankh. des Herzens." p. 193, par. 7.

§ *Dubl. Journ. Med. Sciences.* 1832. 1 Bd.

‖ "Dissert. Tubercle of Myocardium." 1901.

Digitized by Google

cheesy degeneration (Townsend's case was of this nature). When diffuse tuberculosis exists it is the result of infiltration of the muscular tissue by the tubercular granulation tissue, which is soon transformed into a homogeneous cheesy mass, composed of a conglomeration of tubercular masses.

Favourite Seat in Myocardium.—Weigert * called attention to the fact that miliary tubercles occurred most commonly in the ventricles.

Generalisation in Tuberculosis of Heart Muscle and Endocardium.

Frequency of Occurrence.—Haberling† made a *résumé* of recent cases (in 1865) and reports twelve cases. Schöffler ‡ went over the same ground in 1873, and Sanger notes nineteen cases of tuberculosis of myocardium in Schöffler's report. Pollak finds twenty-seven cases. Kaufmann § says he found the smaller nodular form so often, he scarcely thought it worth referring to.

Hartog ‖ finds in modern literature, aside from the cases I have noted above, only nine cases, and adds one of his own in which the complete autopsy is reported.

Diagnosis.—The diagnosis was made but once during life, and then only some cardiac lesion was suspected, but not its exact nature.

At the autopsy, or should the diagnosis of tuberculous tumour become possible in life, only a gumma could be mistaken for a large cheesy mass. In the smaller nodular form accompanying general miliary tuberculosis, careful investigation at the autopsy and careful staining will furnish sufficient evidence. If the large cheesy mass is present, this usually accompanies general tuberculosis elsewhere in the body (mostly in the lungs, often in the bones (vertebræ) and joints, commonly in the glands). Here the presence of the general tuberculosis would make the diagnosis as to the nature of the cheesy mass quite easy.

Age and Sex.—Infants generally furnish the miliary form already referred to. All ages may be affected. Old age furnishes the larger number of cases of cheesy nodules. Data are insufficient for the determination of the influence of sex, though the male sex predominates in the cases noted.

Favourite Seat.—While the favourite seat in acute general miliary tuberculosis is the *conus arteriosus* of the right side, the great papillary muscle, and less frequently the endocardium of the ventricles, it is different in the small nodular form, which is not general, in which the ventricles are the favourite seat; and different again in the larger masses (caseous form), in which the auricles or the junction of auricles and ventricles and the ventricles (often near the septum) are the seat of predilection.

At times, in cases in which pericarditis co-exists, the tumour may have gone by contiguity over to the myocardium, and thus be seated anywhere.

---

* *Deutsche med. Wochenschr.* 1883. No. 24.

† "Diss. Tuberc. Myocarditis." Breslau, 1865.

‡ *Zeitschrift f. klin. Medicin*, "Über Tuberkulose des Herz-Muskels." Bd. 21.

§ *Berl. klin. Wochenschrift.* 1897. No. 31.

‖ *Loc. cit.*

Digitized by Google

Mode of Infection.—The blood-vessels are most probably the main avenues of infection. The large percentage of cases in which tubercular nodules are found in the myocardium in connection with acute general miliary tuberculosis, and in which the blood-vessels are the avenues of infection, would certainly lead us to infer the same avenue in the less frequent cases. Again, the fact that the two varieties occur in different situations, or because in the one case we deal with minute, almost invisible tumours, is no argument against the probable identical source of infection in all.

Clinical History.—Although two cases gave rise to indefinite local symptoms, no clinical picture worthy of attention can yet be adduced.

Tubercular Pericarditis.

The incentive to this paper was largely given by an interesting case of this kind seen by me some years ago. Fortunately a still clearer and more beautiful specimen was placed at my disposal by Professor Bostroem, of Giessen, to whose kind assistance I am indebted for the opportunity of studying a magnificently preserved specimen, of which I append a photograph. A second case, whose history is briefly told below, is of importance as supporting a now generally accepted view as to the origin of the infection in tubercular pericarditis.

Case 1.—A man thirty-six years of age entered the Surgical Clinic at Giessen for a right-sided empyema. Nothing pointed to cardiac involvement during life, and beyond probable large heart area (presumably recognised), no minutes of any suspicion of tuberculosis are found.

At the autopsy the pericardial sac was enormously distended. The distended sac was 24 cm. broad and 20 cm. long. The outer parietal pericardial sac was covered with numerous grey miliary nodules, which extended and covered a limited portion of the right pleura. The sac contained considerable thick bloody fluid; 1½ litres were emptied. The visceral pericardium was completely covered with a series of layers of greyish cheesy nodules, of fibrous tissue mixed with serum and pus, and partly formed fibrinous layers. The whole formed a firm mass which was firmly adherent to the heart. The most striking feature was the layer of cheesy nodules, 1 mm. in diameter, which was next to the heart muscle and formed a clearly distinguishable layer. Over this lay a layer, 1 cm. in thickness, of young tuberculous granulation tissue, and upon this came the layer of fibrine mixed with sero-pus and blood. The photogravure accompanying paper shows layers quite distinctly.

The right side, in the region of from the fourth to the sixth rib, was the seat of a sharply localised tuberculous sacculated empyema. Lung and spleen contained a few tubercles. The tracheal and bronchial glands were enlarged and cheesy, and the seat of anthracosis (the man was a miner).

There is no doubt that the cheesy bronchial and tracheal glands were the starting points of the pericarditis, and that the latter had been excited by extension of the tubercular glandular tissue.

Digitized by Google

Tuberculosis of the Heart.

the parietal layer of pericardium shown about half its natural

TO ILLUSTRATE MEMOIR ON TUBERCULOSIS OF THE HEART BY PROF H NEWTON HEINEMAN M D

Digitized by Google

Digitized by Google

Case 2. Tuberculous Adhesive Pericarditis.—Woman, 68 years old, died of marasmus.

At the autopsy, atrophy and anæmia of nearly all organs were found. The lungs were free from tuberculosis, save for a few small old fibrous nodules. In no other organs was any tuberculosis found, save in the following. The heart sac was totally adherent, and between the heart surface and the adherent pericardial wall greyish-yellowish tuberculous nodules covered the entire surface of the heart. The heart was atrophied. The cervical glands and the bronchial glands at the tracheal bifurcation were the seat of tubercular cheesy degeneration, which had destroyed much of the gland tissue. The gland tissue was pigmented. The tubercular pericarditis was the sequence of the penetration of the cheesy degenerated bronchial glands into the pericardial sac, which was adherent to the heart by tubercular pigmented tissue.

Etiology.—While spontaneous tubercular pericarditis is not denied as a possibility, its probability is considered more than doubtful. The more thorough the investigation of a given case, the fewer will be the idiopathic cases, and the greater the number of cases in which some preceding infection will be found.

Zenker,* years ago, pointed out the fact that in fifty-four cases of œsophageal ulcers caused by diseased lymph glands, ten cases of pericardial adhesion were found. In 1879 he confirmed the relation between diseased mediastinal and bronchial glands and the pericarditis. Kast reports a case of Zahn's, of purulent pericarditis, with perforation of cheesy bronchial glands into the pericardial sac.

Weigert† has also called attention to the fact that the serous membranes generally, when infected with tuberculosis, acquire the disease by contiguity from a neighbouring organ.

Special attention is called, not so much to the bronchial glands as to the set of small mediastinal glands seated below the bifurcation of the trachea, and above the place of folding over of the pericardial sac, as the source of irritation of the tubercular pericarditis. These glands first become tubercular and then invade the pericardium by extension.

Kast quotes cases of Baümler and Schottelius confirming this mode of infection.

Age and Sex.—Nothing is characteristic in tubercular pericarditis as to age and sex, except that even old persons may have this form of pericarditis.

Form.—The usual characteristics of tubercular pericarditis are too well known to require repetition here. As an unusual phenomenon we find the arrangement of cheesy nodules in a complete enveloping layer, as evidenced in Case No. 1 referred to by me. An additional interesting form is the pigmentation in Case II.

Clinical History.—While a larger number of cases of tubercular pericarditis are recognised during life, they are rarely, if ever, of the class of which we speak here. The possibility that even in advanced life, if ever cheesy glands have existed, such a sequence is possible, should be borne in mind.

---

* Zenker and v. Ziemssen: "Krankh. d. Œsophagus," v. Ziemssen's "Cycl." Bd. VII.

† *Deutsche med. Wochenschrift.* 1882. *Virchow Archiv.*, 96.

Digitized by Google

Staining Methods.

Wechsberg * gives two excellent staining methods which he used in the laboratory of Geheimrat Weigert, and in that of Geheimrat Ehrlich. In his second method, he succeeded with a general tissue stain, including cell nuclei and protoplasm, connective tissue and elastic fibres, without clouding the bacillus stain in the least.

It must be remembered that Wechsberg worked with the very fresh, comparatively delicate tissues of recently-killed infected rabbits.

For fixing and hardening, alcohol was generally employed, only the eyes were hardened for one or two days in 10 per cent. formol. Some preparations were fixed in Flemming's solution, and coloured with safranin, to make the proliferating nuclear appearances more distinct.

The tissues were subsequently embedded in the usual manner, partly in celloidin and partly in paraffin.

Staining method—

(A.) 1. Section staining for one hour at 37° in thermostat in carbolfuchsin.
2. Differentiation in muriated and 70 per cent. alcohol.
These 1 and 2 were bacilli stains.
3. Staining with Delafield and van Gieson haematoxylin in usual manner (van Gieson).
4. Absolute alcohol, xylol, balsam.

In such sections the bacilli were dark red, the cell nuclei blue or blue-violet, the cell protoplasm yellowish-brown, the connective tissue pale red.

(B.) 1. Staining of section for an hour at 37° in carbolfuchsin.
2. Differentiation in muriated and 70 per cent. alcohol.
1 and 2 are bacilli stains.
3. Section staining for one hour in Weigert's solution for colouring elastic fibres.
4. Rinsing in 96 per cent. alcohol.
3 and 4 are elastic fibre stains.
5. After colouring in Lithion carmine, and washing with muriated alcohol. (For contrasting colouration, nuclei and protoplasm colouring.)
6. Absolute alcohol, xylol, balsam.

In these preparations the bacilli were dark red with a tinge of violet, the elastic fibres were dark blue, the remaining tissues of the contrasting colouration were light red, with more darkly-stained nuclei. Care must be taken not to make the Lithion carmine staining too intense, when the red bacilli staining was always clearly recognisable, against the violet-tinted red ground-work.

Hartog * mentions the following process applied to a large tumour mass of

* *Loc. cit.*

Digitized by Google

tubercular tissue from the human subject. A piece was hardened in 10 per cent. formol, then placed in 96 per cent. alcohol, and finally in *absolute* alcohol. The pieces were then put in alcohol-ether, and embedded in celloidin. Sections of 0,015 mm. thickness were now stained after the von Gieson method.

Finally coloured with the Weigert stain for elastic fibres.

LARYNXTUBERKULOSE UND GRAVIDITÄT.

VON DR. A. KUTTNER, *Berlin.*

DER Einfluss den die Gravidität auf die Larynxtuberkulose ausübt, ist bisher nirgends und niemals eingehend gewürdigt worden. Es finden sich in der Literatur 7 Fälle, von 4 Autoren mitgetheilt, die als casuistische Belege geeignet sind, das Krankheitsbild, das aus dem Zusammentreffen von Schwangerschaft und Kehlkopftuberkulose resultirt, zu illustriren, man hat es aber bisher immer versäumt, aus einer Zusammenfassung und kritischen Würdigung der verschiedenseitigen Erfahrungen ein Regulator abzuleiten, das wenn auch nicht für jeden einzelnen Fall, so doch principiell gültige Verhaltungsmaassregeln an die Hand geben kann. Dieser Mangel hat sicher manches Opfer gekostet, das bei besserer Klärung der Situation vielleicht hätte vermieden werden können. Zweck dieses Vortrages ist es, eine Anregung zu bieten, wie diesem Übelstand nach Thunlichkeit abgeholfen werden kann.

Das Material, den Verfasser zusammenzutragen in der Lage war, umfasst 14 gut beschriebene Fälle und etwa 10–12 weitere Fälle, von denen sich genauere Details nicht beibringen liessen. Von diesen 14 Fällen sind 7 bereits in der Literatur niedergelegt; alle übrigen Fälle entstammen zum Theil dem Beobachtungsmaterial des Verfassers, zum Theil einer Enquête, die Verfasser bei einer Anzahl von Collegen, die über umfassende Erfahrung verfügen, angestellt hat. Zu besonderem Dank fühlt sich Verfasser verpflichtet gegen die Herren B. Fränkel und Gusserow für die freundliche Überlassung ihres Materials.

Die Ergebnisse dieser Untersuchungen waren folgende: Eine hereditäre Veranlagung liess sich durchaus nicht in allen Fällen nachweisen. Bei 3 Frauen war die wohl in allen Fällen primäre Lungerkrankung schon vor Beginn der Schwangerschaft deutlich ansgesprochen; in den 11 anderen Fällen war von Seiten der Lungen keine oder nur eine minimale Erkrankung nachweisbar. Die Kehlkopferkrankung bestand einmal schon vor der Conception, 2 mal trat sie im 6. Monat auf, 11 mal in der 1. Hälfte der Gravidität. Erst- und Mehrgebährende sind der Erkrankung in gleicher Weise ausgesetzt. Eine Frau die früher an Kehlkopftuberkulose gelitten hatte, mit äusserst geringer Betheiligung der Lungen, die nach ihrer Ausheilung 3 Jahre lang ganz frei von jeder Beschwerde geblieben war, erkrankte sofort nach dem Eintritt der Schwangerschaft aufs Neue.

Digitized by Google

Ganz ausgetragen wurde anscheinend kein Kind: 3 wurden im 9., 8 im 8., 3 im 7. Monat geboren. Alle Kinder kamen lebend zur Welt, bei 5 fehlte jede Nachricht über ihr weiteres Schicksal; von 2 Kindern konnte Verfasser in Erfahrung bringen, dass sie noch leben (das eine 7 Monat alt an schwerem Keuchhusten leidend, das 2. fast 2 Jahre alt, gesund); 7 Kinder sind gestorben, zum Theil ummittelbar nach der Geburt, spätestens 3 Wochen alt; das sind von den 9 Kindern die hier in Betracht kommen 77–78%.

Die 14 Frauen, über die genauere Berichte vorliegen, sind alle ausnahmslos gestorben, zum Theil unmittelbar nach der Entbindung, spätestens aber 2 Monate nachher.

Die nicht genau registrirten Fälle zeigten fast durchgängig dasselbe Krankheitsbild; hin und wieder schein aber eine Frau mit dem Leben darangekommen zu sein und sich wenigstens einigermassen nach der Entbindung erholt zu haben. Es wird Sache einer späterer Forschung sein, festzustellen, wie gross die Zahl derer ist, die die Tähmisse einer durch Larynxtuberkulose complicirten Schwangerschaft und Entbindung überstehen.

Die in der sonst üblichen Weise vorgenommenen Localbehandlung per vias naturales ist durchaus erfolglos gewesen; eine spontane Besserung, so lange die Schwangerschaft besteht, dürfte wohl angeschlossen sein.

Aus den eben skizzirten Erfahrungen glaubt Verfasser folgende Schlussfolgerungen ableiten zu dürfen.

Bei Frauen, deren Befinden hoffnungslos ist, soll man die Larynxtuberkulose nur in der üblichen Weise local behandeln ev. die Tracheotomie vornehmen.

Bei Frauen, deren Allgemeinbefinden günstig ist, darf man, so lange die Kehlkopferkrankung ganz geringfügig ist (kleine Erosionen, ein circumscriptes Ulcus) sich abwartend verhalten. Sobald sich Infiltrate bilden oder die Erkrankung sich anschickt, diffus zu werden, soll man die Patientin über die Gefahren ihres Zustandes unterrichten und nach erlangter Einwilligung so schnell als möglich die Tracheotomie vornehmen und wenn diese nicht in wenigen Tagen günstig wirkt, den künstlichen Abort einleiten.

Je früher die Schwangerschaft unterbrochen wird, desto günstiger liegen die Chancen für die Mutter, weil die Anstrengung für die Mutter eine so geringer ist, je kleiner die Frucht ist. Auch der Blutverlust pflegt beim künstlichen Abort verhältnissmässig gering zu sein. Von 7. Schwangerschaftmonat ab verschlechtern sich die Aussichten für die Mutter, weil der Anstrengung bei dem Geburtsakt eine starke Erschöpfung zu folgen pflegt.

Es ist ratsam bei vorgeschrittener Kehlkopferkrankung vor der Entbindung die Tracheotomie vorzunehmen oder wenigstens zu ihrer Ausführung sich immer bereit zu halten, um einer plötzlichen Erstickung während des Geburtsaktes vorbeugen zu können.

Digitized by Google

TUBERCULOUS LARYNGITIS AND PREGNANCY.

By Dr. A. KUTTNER, *Berlin.*

THE influence which pregnancy exercises on tuberculous laryngitis has, until now, nowhere or at any time been thoroughly estimated. In the literature, contributed by four authors, are to be found seven cases which can be used to exemplify the clinical picture which results from the combination of pregnancy and tuberculosis of the larynx. There has, however, so far been a failure to deduce from the comparison and critical study of the different cases any conclusions which, if not applicable to every single case, can yet furnish general rules for guidance. This want has surely cost many a sacrifice which could have perhaps been avoided by a better appreciation of the situation.

The purpose of this memoir is to offer a suggestion as to how this bad state of things can be practically remedied.

The material which the writer was able to bring to bear on the situation consists of fourteen minutely described cases, and about ten or twelve cases of which more exact details could not be brought forward. Of these fourteen cases seven have already appeared in medical literature; all the remaining data are based partly on the observations of the writer, and partly on inquiries which the lecturer has instituted amongst a large number of colleagues of wide experience. The writer feels himself specially indebted to Herrn B. Fränkel and Gusseron for kindly allowing reference to their observations.

The results of this investigation were as follows:—A hereditary tendency is certainly not authenticated in all cases. With three women primary lung disease was clearly indicated before the beginning of pregnancy; in the eleven other cases there was no disease, or only a minimum, shown in the lungs. Laryngeal disease existed in one case before conception, in two cases it appeared in the sixth month, in eleven cases in the first half of gestation. Both in the case of first and later conceptions the disease appeared in the same way. One woman who had suffered earlier from laryngeal tuberculosis, with exceedingly slight affection of the lungs, and who remained quite free from every disorder for fully three years after her recovery, became ill again immediately after the onset of pregnancy.

No child was carried quite to full time; three were born in the ninth month, eight in the eighth month, three in the seventh. All the children were born alive. As to the subsequent fate of five of these information is wanting; of two the writer can testify that they still live (one seven months old suffering from severe whooping cough; the second, nearly two years old, was healthy). Seven children died, some immediately after birth, some, at the latest, at three weeks old, thus making out of the nine children here considered a mortality of 77 to 78 per cent.

The fourteen women, about whom more exact details are to hand, all

Digitized by Google

without exception, died, some immediately after parturition and some, at the latest, two months afterwards.

Of the cases not so exactly recorded almost all recovered, at least partly, after parturition. It will be a matter for a later inquiry to settle exactly what proportion of cases survive the period of a pregnancy complicated by the dangers of tuberculous laryngitis.

The treatment usually undertaken locally has been entirely unsuccessful.

The author thinks he may draw from the examples sketched above the following deductions :

1. In women whose recovery is hopeless tuberculous laryngitis can only be treated by the usual local remedies or by performing tracheotomy.
2. In women whose general health is favourable one may, so long as the affection of the larynx is insignificant (slight erosion or circumscribed ulceration), pursue an expectant plan. So soon as symptoms of infiltration show themselves, or the disease extends and becomes diffuse, one should acquaint the patient with the danger of her condition, and, after obtaining her consent, should perform tracheotomy as soon as possible, and if this does not act favourably in a few days, should induce premature labour.

The earlier the pregnancy is interrupted so much the more favourable are the chances for the mother, because the strain on the mother is less the smaller the fœtus is. Besides, the loss of blood is usually less in a favourable abortion. From the seventh month of pregnancy onwards the prospects for the mother are worse, because complete exhaustion usually follows the strain of parturition.

It is advisable to perform tracheotomy in advanced laryngeal disease before parturition, or at least to hold oneself always in readiness to carry it out, in order to be able to obviate sudden asphyxia during the act of birth.

THE PRINCIPLES OF TREATMENT OF TUBERCULOUS LARYNGITIS.

By StCLAIR THOMSON, M.D., M.R.C.P. Lond., F.R.C.S. Eng.,

Physician to the Throat Hospital, Golden Square; Surgeon to the Royal Ear Hospital, London.

The statistics of the pathological department of the Brompton Consumption Hospital show that the larynx is affected in over fifty per cent. of the cases which succumb to pulmonary tuberculosis. As 70,000 persons die annually in the United Kingdom from this disease, at least 35,000 of them would have claimed our help in diminishing their sufferings from tuberculosis of the larynx. The statistics of averages warrant us in saying that there are

Digitized by Google

in this country at least 75,000 who require our aid in arresting or easing the progress of tuberculosis of the larynx. The widespread character of this disease is therefore in itself a claim upon our attention; and when we remember the long drawn-out sufferings which may accompany it, and the youth of the majority of its victims, our humanity is keenly stimulated on their behalf.

The moment seems opportune for briefly reviewing the principles which may guide us in the treatment of tuberculosis of the larynx, for not only must we readjust older views to the modern light which has come upon the scene, but such an occasion as the present congress rarely occurs for supplementing the experience of the laryngologist by that of the general physician and the pathologist. That this review is very necessary has been impressed upon me by the perusal of a large number of the most recent text-books on laryngology, few of which contain any reference to the treatment of laryngeal tuberculosis by modern hygienic methods. The frame of mind of many laryngologists is reflected in a recent paper by Dr. Johann Sendziak, in which he makes mention of the "rational—that is, the surgical—treatment" * of this disease, as if any method of treatment short of surgical was not worthy of being denominated as reasonable, and as if hygiene and rest were of no avail, and the *vis medicatrix naturæ* a myth.

Our principles of treatment are guided by clinical experience, but, when available, are based on pathological knowledge. The pathology of tuberculous laryngitis is rendered difficult by the complexity of the anatomical arrangement of the larynx. The varieties in the structure of the mucous membrane and submucosa, the functions it performs, the proximity of tendons, ligaments, muscles, cartilages and joints, the disposition of lymphatics and vessels, the occasional movements required in deglutition and the constant rhythmic action of the vocal cords in respiration, are all points which have to be taken into consideration. While the morbid histology of tuberculosis can be so readily studied in the larynx that Virchow recommended it as one of the best opportunities for observing the process, yet the complicated nature of the larynx renders an investigation of the anatomical conditions an equally important part of our task.

Tuberculous affections of the larynx have been classified under four categories:

(*a*) Superficial ulceration commencing from the surface;
(*b*) Infiltration, followed by
(*c*) Ulceration; and
(*d*) Tumour formation, or tuberculoma.

This classification is of course somewhat arbitrary. It is seldom that two or more of these forms are not combined when a case first presents itself. As there is little doubt that in the large majority of cases infiltration precedes every other process, it is deserving of particular study as to its situation. It

* 'Journal of Laryngology,' May, 1901.

Digitized by Google

commences in the subepithelial layer, and when it takes place in regions where the mucous membrane is closely adherent to deeper tissue, and particularly to cartilage—as in the epiglottis, vocal processes, and arytenoids —it is very apt to spread to deeper parts, leading to perichondritis and necrosis of cartilage. Although the mucous membrane of the vocal cords is closely attached to the underlying tissue, the absence of subjacent cartilage renders infection of this part of the larynx a less rapidly destructive process. On the ventricular bands there is still less danger of immediate spread to adjacent cartilage.

Of all the various situations in the larynx the most frequently attacked is that of the arytenoids and the neighbouring inter-arytenoid space. Lake found this part affected twice as often as the vocal cords, and three times as often as the epiglottis and ventricular bands.*

In the early stages of such cases the vocal cords not only show a want of tension, but careful inspection will show that their movements are impaired both in adduction and in abduction. This tendency to remain in the natural cadaveric position (i.e., the position of rest), the inter-arytenoid thickening, and the consequent dysphonia or aphonia, have inclined W. Fowler to look upon tuberculous laryngitis as chiefly a joint disease. He supports his view by the record of between forty and fifty autopsies of tubercular laryngitis, and as his knowledge as a laryngologist helped to render these examinations very complete, I think the results deserve careful consideration. "In every case," he writes, "the greatest seat of the mischief was in the immediate neighbourhood of the crico-arytenoid joint, and the joint itself was always implicated. The deepest part of the ulcer, when ulceration existed, was always immediately in front of the joint, and the joint not only communicated with the floor of the ulcer, but was also more or less disorganised. In many cases the arytenoid was a loose piece of dead cartilage." †

The pathology of laryngeal tuberculosis requires still further study, but in any case we seem warranted in assuming that, as in other parts of the body, the first process is one of infiltration. Universal clinical experience and pathological observations concord in establishing the fact that in a large majority of cases this infiltration first takes place in or about the arytenoid joints. Other parts are occasionally attacked primarily; the epiglottis less frequently than any other.

Leaving now for a moment the pathological aspect of the subject, let us consider it from the result of treatment. Writing in 1880, Morell Mackenzie observed, "It is not certain that any cases ever recover" (p. 383), and he states that he only knew of four in which he had reason to believe that the disease was entirely arrested. ‡

This view has been somewhat modified in the succeeding twenty-one years by the work of Moritz Schmidt, Krause, Heryng and others. Their

* "Laryngeal Phthisis," London, 1901.

† "Intercolonial Medical Journal of Australasia," October 20, 1898.

‡ "Diseases of the Throat and Nose," vol. i., p. 383.

Digitized by Google

work has, unfortunately, diverted attention too exclusively to the possibility of exterminating the disease from the larynx by knife and caustic. Recoveries have, indeed, been claimed under various treatments, but we must remember that arrest will take place in the larynx as elsewhere without any local treatment whatever. When reaction and resistance of neighbouring tissues are sufficiently vigorous, the advance of infection is checked by the fibroid change, which is the natural and desirable process of cure. In many cases the recovery is deceptive; partial cicatrisation of an ulcer may take place in one part, or retrogression of an infiltration occur in the region visible in the mirror, while the process may be spreading in the depths of the tissues, or in such parts as the ventricles of Morgagni and the subglottic region. Besides, the foreshortened image we see in the mirror is a very unsatisfactory picture of the posterior laryngeal wall—the most important region in tuberculosis—and is always inadequate as regards the parts lying below the cords. Every one who performs a laryngo-fissure, or opens a larynx on the post-mortem table, is prepared to find disease invariably more extensive than it appeared in the laryngoscope.

But what remains to us of all the various methods of local treatment which have from time to time been vaunted as curative of laryngeal tuberculosis? Their very number is eloquent of their inefficiency, and although some cases may have recovered under treatment, and many may have been locally relieved, yet we need hardly stop to consider whether the various sprays, pigments, insufflations, submucous injections, or intratracheal injections, had more than an alleviative effect, or whether, in the majority of cases, the irritation and reaction they produced did not far counterbalance any possibility of good.

None of the numerous methods which have from time to time secured some attention have ever appeared to me sufficiently rational to make them worthy of an extended trial. On the other hand, their disadvantages and uncertainties were only too apparent. I have therefore been compelled to appeal to the experience of others on this matter, and in doing so will only refer to what we may term the lactic acid and the surgical methods of treatment.

Applications of lactic acid to the tuberculous larynx have obtained such a vogue in the last ten or twelve years that the method has been applied *à tort et à travers*, practitioners in many cases persevering with it while the patient was being prevented, through its effects, from improving generally, or even steadily deteriorating in health. In many cases I have known of its being applied over unbroken mucous membrane, covering deep infiltrations, or evident perichondritis, the surgeon apparently not stopping to ask himself how this superficial caustic could effect these deep processes, or do more than distress the patient and hurry on the progress of the disease. And now Freudenthal, who used it freely, states frankly that "it ought to be dispensed with as antiquated and barbarous torture of the patients." *

* *Journ. of the Amer. Med. Assocn.*, 16 March, 1901.

Digitized by Google

In 1899 Freudenthal subjected twenty-nine cases to surgical treatment without being able to record one single cure.* He then treated his cases of tuberculous laryngitis without curettage, and after a year's observations he wrote, "I believe my patients are just as well and perhaps better off than they would have been with the operation."†

The extensive and trustworthy experience of Jonathan Wright has led him to the following statement: "The permanent radical cure of the local lesion of tubercular laryngitis is not materially hastened by the various methods of treatment in any but an insignificant number of cases."

That a certain number of apparently permanent cures have been affected is undoubted. I have myself verified such a case both before and after treatment, which was shown by Dr. Lack to the Laryngological Society of London,‡ but the chief point to realise is that even the most enthusiastic supporters of surgical treatment of tuberculous laryngitis admit themselves that the majority of cases are unsuitable even for attempting operative measures. We must also remember that in this small minority of cases the method is painful and distressing; it cannot but react unfavourably on any general condition; and the result is extremely doubtful.

It seems to me that the treatment of the last decade has been based too exclusively on the bacillus as the one and only etiological factor, and that due regard has not been given to more general considerations.

In indicating the slight and unsatisfactory results which have been gained from the direct treatment of laryngeal tuberculosis I must be understood as only deprecating much of the treatment in so far as it has been regarded as affecting a local cure. Where the progress of the disease—in the lungs and in the larynx—is not stimulated by local interference then many measures are available for symptomatic treatment, and we are well equipped nowadays for soothing laryngeal irritation and cough, easing pain, facilitating swallowing, and thus contributing to the general treatment and the possibility of cure.

We must look elsewhere at present than to surgical measures for a prospect of progress in the treatment of tuberculosis of the larynx. This progress is ready to hand in the making of an earlier diagnosis of local infection. The present is hardly the occasion, even if time permitted, for me to enlarge on the symptoms of the early diagnosis of laryngeal tuberculosis. Besides, the most detailed description of the laryngoscopic appearances could hardly portray a condition which would be recognised by any but an expert, so slight are the early changes and so variously are they combined. "In general," says Grünwald, "it may be said that it is impossible to teach anyone theoretically how to make a diagnosis from the picture in any given case, because, in order to arrive at a decision, one must first learn the development of many successive pictures by long personal observation. Not the picture of to-day, but that of yesterday, and that of to-morrow, must

* *Philadelphia Med. Journ.*, 25 March, 1899.
† *Medical News*, New York, 19 Jan., 1901,
‡ 'Trans. Laryngol. Soc.,' London,

Digitized by Google

decide for or against laryngeal tuberculosis." * But it is not only from the laryngoscopic appearances that a diagnosis of early local tubercular infiltration, or of even pre-tubercular laryngitis, can be made. We must make a careful and thorough examination of the entire body, and pay careful attention to such symptoms as anæmia, anorexia, dyspepsia, loss of weight and strength, hurried pulse, and evening rise of temperature. The previous history of the patient, particularly in regard to hæmoptysis and pleurisy, must be taken into consideration, and the family history should not be forgotten. There are many other indications of early tuberculosis, and these, together with the indications for the employment of tuberculin as a diagnostic test, I must at present leave out of consideration. In this way evidence can often be obtained which will complete the diagnosis of a laryngeal condition which might otherwise be treated as a simple catarrh. In the absence of positive confirmatory symptoms, and of other adequate explanation of laryngeal symptoms, we must treat suspicious cases by measures that we know now will avert a condition which, once well established, is almost always incurable. In doing this we are but working along the lines and making the same plea for early diagnosis which has been so forcibly advanced in recent years in the subject of pulmonary tuberculosis.

Once the early diagnosis is made the treatment is exactly the same as that now employed in pulmonary phthisis—the sanatorium treatment in what should practically be the open air, with rest, hygienic surroundings, and good food. To this must be added, more or less, strict insistence on voice rest. This is found to be beneficial in many cases, even when the larynx is not affected. It must be much more so in laryngeal cases, when we realise that in the majority of instances the focus starts near or in the crico-arytenoid joints.

The treatment of catarrhal or obstructive affections of the nose and throat, and of any intercurrent conditions of the larynx, must, of course, receive careful and suitable treatment, and it is, therefore, very desirable that those in medical charge of sanatoria should be skilled in practical laryngoscopy. But the important principle to bear in mind is *primum non nocere*, for even a clumsy examination of the throat may produce more irritation and harm than any treatment can counterbalance.

Briefly recapitulated, the principles to bear in mind in tuberculosis of the larynx are as follows :—

1. Pathology and clinical experience show that in the majority of cases the focus of infection is near or in the crico-arytenoid joint.

2. Many cases only present themselves at a stage when the possibility of effecting a cure by local measures is quite untenable.

3. The principle of *primum non nocere* should be constantly kept before us, as many measures which have been tried in this affection have only distressed the patient and hastened the disease.

4. In the light of present knowledge and therapeutic resources, the most rational principle is to attempt to make an early diagnosis of the disease while

* "Atlas and Abstract of the Diseases of the Larynx," 1898.

Digitized by Google

in an incipient stage. Any persistent or suspicious laryngeal catarrh should be treated seriously on even a presumptive diagnosis.

5. Once diagnosed, the patient should be treated on the principles laid down in the modern method of sanatorium treatment.

6. Symptomatic treatment should be directed to any irritative, catarrhal, or obstructive condition of the air passages.

7. In addition, silence should be enjoined, the disuse of the voice being proportionate to the degree in which the focus of infiltration approaches or interferes with the arytenoid joint.

8. In cases where the situation or extent of disease do not warrant an expectation of complete arrest of the process, treatment should be symptomatic, and in many such cases the sanatorium treatment is uncalled for.

TUBERCULOSIS OF THE EYE.

By ALLEN T. HAIGHT, M.D., *Chicago, U.S.A.*

The Paper begins with the history of Tuberculosis in the various parts of the eye; treats of ocular disease as possibly the only and earliest manifestation of tubercular affection; gives the ages in which the local disease may occur, and quotes numerous authorities and conclusions; tabulates the number of published cases; gives the countries in which they occur; report of cases; treats of hereditary and primary Tuberculosis of the eye, and concludes that ocular tuberculosis is due to two causes; urges early enucleation of the affected organ, with the belief that such procedure saves many lives.

Tubercular infection, and particularly primary infection of the eye, is no longer a question, and not uncommon, as was supposed even ten years ago.

As early as 1808, Autenrieth anatomically described tubercle of the choroid, and McKenzie, of Glasgow, nearly three-quarters of a century ago, described tubercular disease of the eyeball, and forecasted that in the near future the profession would recognise a subsequent general infection; but this prophetic assertion was passed unnoticed by all pathologists; even the great Rokitansky, in his list of 14,000 post-mortem examinations, did not mention a single case of ocular tuberculosis.

In 1855 Edward Jäger discovered the presence of tuberculosis in the choroid, by means of the ophthalmoscope.

In 1858 Manz made ophthalmoscopic examinations, and found tubercles in the choroid, and, later, Cohnheim demonstrated the importance of their appearance in acute miliary tuberculosis, especially in tubercular meningitis.

In 1865, Virchow, in his "Krankhaften Geschwülste," first defined tubercular pathology as a neoplasm, which takes its origin from the connective tissue in the form of nodules, consisting of closely packed cells, of a finely granular structure. They develop by regular gradation from simple cells, and

Digitized by Google

are thus shown to be of organic formation, which Virchow regarded as a special form of cell formation.

In 1867 von Graefe and Leber gave exact ophthalmoscopic pictures. Generally, both eyes were affected, the tuberculous deposits being found in region of the macula and papilla.

In the same year Hjort first found tubercle in the chiasm. In 1869 Gradenigo published the first case on record of miliary tubercle of the iris. In 1873 Perles reported the first case of miliary tuberculosis in the retina. In 1874 Koester first published a local case of tubercular conjunctiva, without tubercular disease in any other part of the body.

Hirschberg brought forward the only case of tubercular disease of the conjunctiva of the globe, reported at the International Congress, held in London in 1881; child, aged four; globar conjunctiva (of left eye) surrounding the cornea entirely converted into a confluent caseous ulcer. After excision, rapid and permanent recovery made.

Before we attempt to describe the way in which tuberculosis manifests itself in the various coats of the eye, and its appendages, we are compelled to first take into consideration the four different points laid down by Ludwig Bach :—

1. Tuberculosis of the eye is by no means a rare affection.
2. All parts of the eye may be attacked by the disease.
3. It plays a particularly important *rôle* in diseases of the uveal tract.
4. The eye diseases may be the only and earliest manifestation of the tubercular infection.

Ophthalmoscopic pictures of tubercular deposits show them as round, whitish, or yellowish-white spots, of varying size. Anatomically, the single deposits are occasionally so small that they are not visible to the naked eye; sometimes they are $2\frac{1}{2}$ mm. in diameter. At first, they advance toward the retina, and, later, encroach on the sclera; the development having its beginning in the choric-capillaris; the uniform blanching of the pigment epithelium produces the yellowish-white spots.

Ocular disease may be the only and the earliest manifestation of tubercular affection, and may appear in various forms, isolated, diffuse. It may resemble an ulcer or trachoma, and is known to affect almost every part of the eye, even the orbit.

Haab has collected eleven cases of primary tuberculosis, and is of the opinion that it attacks the tunics of the eye and its appendages in the following proportion: palpebral conjunctiva (lid), 60 per cent.; iris, 30 per cent.; bulbar conjunctiva and choroid, 10 per cent.

From the comparative study of recorded cases by Eyre, it is observed that the age limits are wide; from a ten-months infant to a thirty-year-old man; the disease is, however, more general at, or soon after, puberty, than at other periods. Females are more liable to this particular tubercular lesion than males, in the proportion of 1·5 to 1. The disease is more frequent in the right eye, the predominance of which is probably caused by the infectious material more often conveyed by the right hand.

Digitized by Google

The iris is the most frequent starting-point of tubercle, from where the disease appears to spread to other tunics with much rapidity.

Tuberculosis of the iris is most frequently met with between the ages of fifteen and thirty; it has, however, been observed even beyond that limit. Just how it occurs has been much discussed; some authors believe that a local inoculation takes place in a healthy subject by way of an abrasion of the conjunctiva. Others believe that the initial lesion is a tuberculous ulcer of the conjunctiva. Van Duyse, of Ghent, believes that tuberculosis of the iris is secondary to a diseased focus elsewhere. Tubercular iritis occasionally suggests nodules in its clinical outworking, although when nodules are present tuberculosis is always to be considered.

Leber agrees with Habnet and Haensell that the small reddish nodules which develop on the iris, and which may disappear spontaneously, to be followed in many instances by the appearance of tubercles, are of a tubercular nature. The clinical appearances of tuberculosis of the iris, however, are not always the same, and there are cases where diagnosis is difficult, particularly when experimental inoculation has failed. Some authors claim that tuberculosis of the iris is a rare disease. For instance, Machek found but two cases of tuberculosis of the iris in 15,000 patients; both of the patients died later of tubercular meningitis. Machek differentiates anatomically three forms of tuberculosis of the iris: (1.) Tuberculous infiltration; (2.) disseminated tubercles; (3.) conglomerated tubercles.

Vignes, of Paris, gives the following conclusions in regard to tubercular iritis: (1.) It is important, from a clinical point of view, to differentiate a tubercular variety of iritis. (2.) This form of inflammation is premonitory of the tubercular nodular eruption, which it may precede by several weeks. (3.) It is characterised by its sub-acute mode of invasion; its evolution is slow and torpid, being marked by faint reactional signs, although in addition there may be dense synechiae, more or less completely obstructing the pupil. (4.) The absence of pathognomonic symptoms renders the diagnosis difficult. (5.) The tendency to spontaneous cure of miliary tuberculosis depends upon the individual resistance, and especially upon the resistance of the iris.

Tubercles in the choroid in primary affection generally occupy the region of the chorio-capillaris and the vascular layer. They are also frequently observed in cases of acute miliary tuberculosis, but they may be present in all forms and stages of tubercular disease.

After a thorough study of eight cases of tuberculosis of the choroid, George Carpenter concludes that tubercles of the choroid are protean in their characteristics, and we must be prepared to find them under various guises. In respect to the microscopical characters of these growths, he says that "although in some cases the blood-vessels are far less blocked with blood-clot, yet in others proliferation of the endothelium is seen in various stages," and he "would offer as a suggestion that the giant cell is formed from the cohesion of these proliferated elements into a single cell-mass." He further says that "the giant cell in the choroidal tubercle is not as has been seen, oval or circular in outline, to the exclusion of other forms, but more often than not it

Digitized by Google

is distinctly elongated, and corresponds in outline to the various shapes assumed by the healthy choroidal vessels as seen under the microscope." He believes that "in many instances it is found lying in the blood-vessel, or in a space in which a vascular wall-remnant can be traced. If, then, it is admitted that the giant cell can be formed from the proliferation of vascular endothelium in a vessel of a certain calibre, there is no reason why the process should not go on equally well in vessels smaller still in connection with the main vessel, and so give rise to the processes of the giant cell and the epithelial elements surrounding it." He, moreover, believes that the small cell infiltration is "in part formed by white corpuscles, and in part by proliferation of the connective tissue cells of the choroid."

Brailey describes a tuberculous mass apparently primary, which sprang from the optic nerve and immediately surrounding choroid, simulating, in some of its clinical features, a glioma.

Wagenmann says that tuberculosis of the choroid can be distinguished from glioma of the retina by the early appearances of inflammatory symptoms, including iritis—phenomena which are not present in this stage of glioma.

Tuberculosis of the conjunctiva generally attacks one eye, though we have seen it manifested in both eyes, and must agree with Fuchs, "that the disease occurs, almost without exception, in young people."

The age at which primary tubercle invades the eye is uncertain, the earliest instance recorded is that of conjunctival tuberculosis in a child of eight months, attending Del Monte's clinic at Naples.

Sattler, in 1891, first classified conjunctival tuberculosis. His system consisted of grouping the various forms of this disease as it is visible in broad clinical characters, to the naked eye. He divided them into five different groups, viz.:—

(1.) Characterised by the presence of small miliary ulcers, which later on may coalesce, generally attacking the palpebral, but sometimes affecting the bulbar conjunctiva.

(2.) Characterised by the presence of greyish or yellowish sub-conjunctival nodules, varying in size, but rarely larger than a hemp seed, not unlike the sago granules of trachoma.

(3.) Characterised by the presence of florid hypertrophied papillæ and rounded outgrowths or granulation tissue, springing from the palpebral conjunctiva, or situated in the fornices, and which soon recur after removal (resembling in many respects the velvety granulations present in a tuberculous arthritis). These granulations are accompanied by edema and thickening of the lids.

(4.) "Lupus" of the conjunctiva, characterised by numerous pedunculated, cockscomb-like excrescences in the fornices, of a jelly-like consistency, often showing more or less extensive ulceration.

(5.) To the above four groups one more should be added, in order to embrace those cases which are characterised by the existence of distinctly pedunculated tumours, having the macroscopic appearances of ordinary papillomata, and also such as those designated by Mitvalsky, as "true polypus

Digitized by Google

of the conjunctiva"—cases where there is no involvement of the subconjunctival tisssue, nor the production of any subjective symptom other than slight inconvenience due to purely mechanical causes.

According to M. Vicasse, of Toulouse, tuberculosis of the conjunctiva may present itself in the form of an ulcer, an infiltration, or a polypus. A clinical diagnosis of the first two forms is easy, especially when there is lupus of the face.

Hirschberg estimated tuberculosis of the conjunctiva at 1 in 6000; Mules, 1 in 30,000; Eyre, 1 in 3000.

Fick believes that the rarity of tuberculosis of the conjunctiva and lachrymal sac is attributed to the chemical action of the tears and to the mechanical washing away of the bacilli. When the passage to the nose is obstructed there is likelihood of a mycotic disease of the sac, the so-called dacrocysto-blenorrhea. He moreover believes that the bacillus tuberculosis does not thrive among staphylococci.

Blenorrhea of the lachrymal sac may be produced either by tubercular ulceration of the canaliculi or by a tubercular disease of the bony walls of the canal, with secondary disease of the mucous membrane.

Bock described a tubercular tumour of the lachrymal sac which appears to be of infectious origin. The patient had tuberculosis of the right elbow-joint, with suppuration, and the author believes that infectious material was carried on the fingers of the left hand to the eye.

Salzer, of Heidelberg, reports a case of tuberculosis of the lachrymal glands in a fifteen-year-old girl. The tumour had been diagnosed as adenoma, and had been extirpated as such, but the miscroscope revealed the true nature of the growth. The tubercles had involved the glandular epithelium.

M. Gourfein, of Geneva, in one series of experiments, introduced a solid tubercular culture into the lachrymal sac of rabbits; in another, the lachrymal gland was removed two or three weeks previous to inoculation. In all cases infection was effected, proving that the lachrymal sac can be inoculated, and that the lachrymal fluid does not diminish the virulence of the tubercle bacillus. Fourfein observed three forms of tuberculosis of lachrymal sac: (1) Chronic abscess in the anterior wall, leading to a fistula, the pus escaping into the nasal duct; (2) formation of fungosities with escape from the puncta of thin yellowish pus, containing bacilli; (3) granulations of the sac, to which attention is called by epiphora, the liquid not ordinarily containing bacilli, but being very irritating to the palpebral and bulbar conjunctiva and producing smart conjunctivitis.

Tubercular infiltration of the cornea is more difficult of recognition than of other parts of the eye, although primary tubercular infiltration of Descemet's membrane is common.

Neese reported a case of tuberculous neoplasm of the ciliary body which was diagnosed as syphilis, and the real nature of the disease was not described until after enucleation.

Lobowski found, upon anatomical examination, extensive tuberculosis of the ciliary body, and of the lower half of the retina. This contradicts the

Digitized by Google

observation of Wagenmann, that in diseases of the ciliary body the bulb is usually soft. From this condition there arose fulminating glaucoma, the retina and choroid remaining relatively intact.

In the retina the process is usually found in the inner layer. Tubercular affection limited to the retina is indeed quite rare. Story and O'Sullivan described a case in a girl, aged twenty-one, who stated that in one night she had lost the sight of the right eye. Ophthalmoscopically this eye exhibited intense optic neuritis, but the disc was very much swollen and of brilliant whiteness. There were also a few white spots in the retina near the macula. After removal a posterior part of retina and around the optic nerve was found a tumour one-third of an inch in diameter, and one-fifth of an inch in depth. For some distance round the retina was detached and a firm homogeneous coagulum lay between it and the choroid.

Microscopical examination revealed tumour of typical tubercular tissue, which passed without a break into the inner layers of the retina. Through the vessels numerous small nodules were scattered, which were tuberculous in nature.

Tuberculosis of the optic nerve is very rare, and may occur as part of a tubercular meningitis in both acute and chronic form.

I have collected 308 published cases of tuberculosis of the eye, and 4 cases of my own, thus making a total of 312 cases. Of this number 121 were in the iris, 96 in the choroid, 57 in the conjunctiva, 13 in the ciliary body, 11 in the cornea, 8 in the lachrymal gland, 4 in the retina, and 2 in the optic nerve.

The countries in which they occurred are:

Germany	53
France	66
England, Scotland and Ireland	48
Russia	29
Italy	11
Belgium and the Netherlands	9
Austria	24
United States	28
Australia	5
Switzerland	15
Spain and Portugal	9
Mexico	4
South American countries	11
Total	312

One hundred and eighteen cases where one eye only was affected, about 60 per cent. were affected in one eye, followed by extension to the other; and about 40 per cent. complete destruction of vision.

Ophthalmic tuberculosis without prehistoric cause, 86; general tuberculosis in 60 per cent. of all cases. Total cases I find reported in primary

Digitized by Google

tuberculosis of the eye, where it could not be found in any other part of the body, 58. Among these cases, 37 are reported due to injury; 21 give no cause; 26 were affected in one eye only—17 in the right, 9 in the left; among those 7 extended from one to the other eye.

In 122 cases hereditary tuberculosis was reported. In 48 there were other ocal manifestations. One hundred and forty-two gave no particular history of cause, outside of the 37, of the 58 due to injury; and if all the cases of primary ophthalmic tuberculosis were sifted down, I feel sure it would show a percentage much higher than we expect from the cause of injury. This I can substantiate by Peters, who calls attention to the fact that in a hospital in Bonn, among 10,000 injured, at least 500 manifested tuberculosis at the site of the injury, and this was more frequently observed in cases of slight injuries, where disturbances in function were comparatively trivial.

From this high percentage I coincide with Peters, that there is no organism which is so likely to take root and grow at the site of an injury as the tubercle bacillus.

He also believes that irritation of the ciliary nerves brings about favourable conditions to colonisation and multiplication of the ubiquitous tubercle bacillus, and asserts that many cases of chronic insidious iridochoroiditis, with blindness and lowered tension following either injury or operation, may rest upon a tuberculous basis.

Another point to which I wish to call your attention is the manifestation of tubercular diseases of the eye, at the early age of eight months, reported from Del Monte's clinic, at Naples, and Eyre's case of ten months. Other reports from one year to thirty induce me to believe that many of those cases of tubercular manifestations in the eye are due to a fall or a stroke received by children, unable to take care of themselves, unbeknown to parents or nurses, and that in many cases it requires several years to develop. In older persons the injury may have occurred at a more advanced age, perhaps from a blow or a fall, the history of which was not always obtained by the practitioner. In this opinion I am sustained by Jessop, who records a case of tubercular ulceration of the ocular conjunctiva, with enlargement of the preauricular gland, in a boy; no previous tubercular history; after an injury to the globe by a fall the eye became bloodshot and the preauricular gland became swollen and painful, and a small ulcer with hardened edges noticed in the conjunctiva. This was treated as a specific lesion, with no good results. Inoculation of a guinea-pig from the ulcer had proved fatal from unmistakable tuberculosis.

Fuchs noted a conjunctival tubercular abscess that was in all probability indirectly caused by a grain of dust that had settled and broken the membrane.

There are also many cases where tubercular manifestations of the eyes are present, yet are not recognised, or passed on account of lack of discovery of bacilli, as in a case reported by Weinbaum, of Gottingen, who found primary tubercle of the iris in a child, whose mother was phthisical. Enucleation was performed; no bacilli were found, but the eyes of a rabbit were inoculated

Digitized by Google

with a small portion of the growth, and severe iridocystitis was produced four weeks later, and the microscope revealed the presence of bacilli in the inoculated eyes. I also believe that the majority of cases of tubercular meningitis are due to ocular tuberculosis, as in the case of Knaggs, of Leeds, who recorded tuberculosis of the iris and suspensory ligament, with tubercular deposit in retina, in a boy aged nine; after enucleation, tubercular meningitis and death occurred about seven weeks later.

Gordon Norrie, before the Copenhagen Medical Society, reported a case of primary tuberculosis of the conjunctiva in one eye of a child four years old. No family history of tubercle, nor any other signs of the disease in the patient. Microscopically, no bacilli were determined, although introduction of small masses of the diseased material into the anterior chamber of a rabbit developed tubercular nodules.

Stephen MacKenzie describes a case of tubercular choroid in a girl, aged four, with no tubercular history. The diagnosis was made as chronic tuberculosis of the choroid, and brain necropsy revealed acute tubercular meningitis.

Machek also had two cases of tubercular iritis that terminated in tubercular meningitis, and of all the cases recorded in literature where ophthalmic tuberculosis was the cause of death, especially where there was no hereditary history, I would estimate that not less than 50 per cent. died of either acute or chronic tubercular meningitis. I believe that before allowing the deadly disease to get a hold, if by timely action we would enucleate the infected organ, we could save many lives. As, for instance, in the case reported by Bronner of a tubercular tumour of the iris in a girl aged fourteen; the growth was removed and found tubercular. Later, the eyeball was removed, and there have been no signs of tuberculosis since, and at that time there was no evidence of it elsewhere.

Also, in a case reported by Swanzy, in a girl, aged two, who presented a small white tumour on the surface of the iris; there were also two other minute tumours on the iris. The diagnosis of tubercle was made, and the eye removed; microscopical examination confirmed the diagnosis. Five months later the child was in good health. There was no tubercular disease in the family history.

I also believe that of the 142 cases that gave no history, at least 75 per cent. were due to injury, and the balance of the cases recorded as primary tuberculosis without cause were also due to injury. A careful tracing in my own four cases—one in the iris, two in the choroid, and one in the conjunctiva—revealed the fact that the primary origin of the disease was injury.

Case I.—A girl, aged eight, of healthy German parentage, with no trace of tuberculosis on either side of the family, was brought to me with the iris of the right eye intensely infiltrated with reddish nodules, which later, under the microscope, proved to be tubercular, as the tubercle bacilli were found after enucleation. Upon careful questioning, the mother remembered that about two years previous a younger brother, while playing with a rattan whip, had struck the girl in the eye, causing conjunctival ecchymosis and swelling. The

Digitized by Google

swelling and discoloration subsided after several weeks, and to all appearance the eye was well and sound. Two years later the patient was brought to me, and I found the eye as above stated. Six months later the entire eye was involved, with complete loss of vision, and upon the consent of the parents I enucleated the eye. It is now nearly two years since the operation, and the child is healthy, and there is no other manifestation of tuberculosis in any part of the body.

Case II.—Girl, aged four, who fourteen months before I saw her slipped from a low iron picket fence, striking the right eye, causing a contused wound of the outer canthus and upper lid. The eye was treated by the family physician, and to all appearance made a perfect recovery. Fourteen months later I was called in consultation on account of lowered vision in the right eye. Upon examination I found several whitish tumours in the choroid, involving the macular region. Vision of the eye was reduced to counting fingers at two feet. Stratified blood clots were found in various parts of the fundus. I made a diagnosis of tubercular choroiditis, and advised enucleation, which was performed. The wound healed kindly, but four months later the child was brought to me with iritis of the left eye, and before enucleation could be performed, the child was taken sick and died of what proved to be tubercular meningitis.

Case III.—A boy, aged fourteen, living in the country, was brought to me with the following history: About a year earlier, while quarrelling with another boy, he was struck between the eyes with a stone, the septum being broken, and the eyes became bloodshot and inflamed. No particular attention was given as regards local treatment at the time. On examination I found the sight of the left eye reduced to one-tenth normal; there was intense optic neuritis, and surrounding the disc were eight or ten circular nodules, yellowish-white at centre, and passing gradually at periphery into the colour of the natural choroid. The surface of several of the choroidal tubercles could be traced by retinal vessels. Ten days later the eye was enucleated. Eight months have passed since the operation, and there is no manifestation of further infection from tuberculosis.

Case IV.—Male child, aged two years, on Christmas Day, while playing with a tin horn, fell and struck the sharp edge of the mouthpiece into the upper right eyelid, cutting the conjunctiva at the upper part of the globe. No particular attention was given the child outside of stopping the bleeding. Eight months later the child was brought to me at the suggestion of the family physician. Upon examination I found flat-topped and closely packed together excrescences on the conjunctiva at the point of injury. The glands were also much swollen. These growths or excrescences were excised, and the microscope showed them to be tubercular in structure. There has been no return of the trouble in the past eight months.

In conclusion, I would say, first, that I am reasonably satisfied from the cases that have been reported, and from my own observation, that at least 75 per cent. of all cases of tuberculosis of the eye are due to two primary causes—either to infection from other parts of the body, or from direct injury

Digitized by Google

to the eye; second, that in primary tuberculosis of the eye, early diagnosis and operation robs death of many of its victims.

LITERATURE CONSULTED AND REFERRED TO ON TUBERCULOSIS OF THE EYE.

Albrand, Zehender's klin. Monatsb. für Augenhk., 1892.
Alexander, Centralbl. f. prakt. Augenh., 1884.
Altabas, Rev. internat. de Bibliographie.
Alvarez, Bull. Soc. Anat. de Paris, 1885.
Amman, Klin. Monatsbl. für Augenhk., 1897.
Angelucci, Gaz. Med. di Roma, 1881.
Armaignac, Ann. d' Ocul., 1897.
Auger, Bull. et mém. Soc. de Chir. de Paris, 1878.
Bach, Münch. med. Wochen., 1895.
Arch. f. Augenh., 1893-4-5.
Barozzi, Gaz. Méd. d'Orient, 1868-9.
Barraquer, Rec. d'Ophth., 1882.
Baas, Arch. f. Augenh., 1893-4.
Batut, Midi Méd. Toulouse, 1892.
Baumgarten, Archiv. f. Ophth., 1878.
Benson, Brit. Med. Jour. Lond., 1885—Tr. Oph. Soc. U.K., 1885.
De Berardinis, Lavori d. Clin. Ocul. d. r. Univ. di Napoli, 1897-8.
Bock, Arch. f. path. Anat., 1883.
Binet, Bull. Clin. Nation. Opht. de l'Hôp. d. Quinze-Vingts, 1883.
Bouchut, Gaz. d. Hôp., Paris, 1868-1869.
Brailey and Edmunds, The Ophth. Review, 1882.
Brailey and Hartley, Brit. Med. Jour. 1887—Tr. Oph. Soc. U.K., 1886.
von Breuning, Med. Chir. Centralbl. Wien., 1883.
Bronner, Trans. Ophth. Soc. United Kingdom, 1898-99.
Bruckner, Arch. f. Ophth., 1880.
Burnett, Arch. Ophth., 1890-1891-1893.
Bush, Virchow's Archiv, 1866.
Cargill, Ophth. Review, 1899.
Carpenter, Illust. M. News, 1889.
Chacon, Gac. Méd., México, 1893.
Cheney, Bost. Med. and Surg. Jour., 1896.
Cheuautais, Jour. de méd de l'ouest, Nantes, 1887, s. I. 135.
Clavelier, Arch. méd. de Toulouse, 1897.
Cofler, Arch. d'Ophth., 1880.
Cohnheim, Virchow's Archives, 1867.
Collica, Accordino, Movimento, Napoli, 1879.
Coppez, Al Shifa, Cairo, 1893.
Corazza, Rev. Clin. di Bologna, 1867.
Cornil, Bull. Acad. de Méd., Paris, 1892.
Coupland, Tr. Path. Soc. Lond., 1873-4.
Csapodi, Pest. med-chir. Presse, 1889.
Dahl, Nord. Med. Ark. Stockholm, 1870.
Davidson, Med. Press and Circ., 1888.
Daxenberger, Wochenschr. f. Therap. u. Hyg. d. Auges, 1898-9.
De Berardinis, Lavori d. Clin. Ocul. d. r. Univ. di Napoli, 1897-8.
Decaisne, Gaz. méd. de Paris, 1897.
Delafield, N.Y. Med. Jour., 1867.
Del Monte, Ann. di Ottal., 1879.
Denig, Arch. f. Augh., 1895.
Denti, Atti d. Ass. med. lomb. 1894.
Deutschmann, Arch. f. Ophthal., 1881.
Deutschmann, Von Graefe's Archiv, Vol. 27, 1881.
Dryardin, J. d. sc. méd. de Lille, 1885.
Dodd, Trans. Ophth. Society of United Kingdom, 1891-92.
Dolard, Arch. d'Ophth., 1895.
Doyne, Tr. Ophth. Soc. U.K., 1890.
Dubrisay, Gaz. d. Hôp., 1875.
Duci, Gazz. Med. ital. lomb., 1888.
Edmunds, Tr. Ophth. Soc. U. Kingdom, 1881-2.
Elschwig, Mitth. d. Ver. d. Aerzte in Steiermark, 1889.
Eperon, Arch. d'Ophth., 1883.

Digitized by Google

Eyre, Trans. Ophth. Soc. of the United Kingdom, 1897–99.
Fage, Ophth. Klin. Stuttg., 1898.
Falchi, Gaz. d. osp., Milano, 1882.
Fall, Ber. d. k. k. Krankenh., Wieden, 1876.
Ferrett, Bull. Clin. Nat. Ophth. de l'Hôp. des Quinze-Vingts, 1885, Vol. III., p. 237.
Fick, Corresp.-bl. für Schweiz. Aerzte, 1892.
Fieuzal, Bull. Clin. Nat. Ophth. de l'Hôp. des Quinze-Vingts, 1883.
Fisher, Ophth. Review, 1899–1900.
Fontan, Rec. d'Ophth., 1886.
Fraenkel, Arch. für Ophth., 1868.
Fuchs, Text-book of Ophthalmology—La Semaine Médicale, 1888.
Galezowski, Rec. d'Ophth., 1897.
Galezowski, Ann. de Dermat. et Syph., Paris, 1897.
Gallemaerts, Recueil d'Ophthalmologie, 1900.
Gallenga, Rassegna de sc. med., 1891.
Gallenga, Gior. d. r. Accad. di Med. di Torino, 1885.
Gasparino, Ann. di. Ottal., 1891.
Gayarin, Vestnik. Oftalmol., Kieff, 1893.
Gayet, Bull. et. mém. soc. franç. d'Ophth., 1885.
Giglio, Sicilia Med., Palermo, 1889.
Gillet de Grandmont, Rec. d' Ophthal., Paris, 1886.
Goldzieher, Centralbl. f. prakt. Augenh., 1893.
Goode, Cincinnati Lancet-Clinic, 1891, vol. 26, p. 415.
Gourfein (Geneva), Ophthal. Rev., 1899.
Gowers, Med. Ophthalmoscopy, 1879.
Gradenigo, Annales d'Oculistique, 1870.
von Graefe, Graefe und Saemisch's Handbuch.
La Grange, Jour. de Méd. de Bordeaux, 1893.
Grant, Caledon. M. J., Glasgow, 1899–1900.
Griesinger, Archiv. f. Ophth., 1868.
Grunert, Arch. f. Augenh., 1896–1897.
Gunn, Ophth. Review, 1895–1899.
Guttmann, Deutsche med. Woch., 1898.
Haab, of Zürich, Annales d'Oculistique, vol. 64.
Haensell, Archiv. f. Ophth., 1879.
Hartley, Ophth. Review, 1867.
Hartridge, Tr. Ophth. Soc. U. Kingdom, 1895.
Heinersdorf, Klin. Monatsb. f. Augenhk., 1898.
von Heppel, Arch. f. Augenh., 1893, 1894.
Herter, Charité-Ann., 1875.
Heymann, of Dresden,
Hill Griffith, Brit. Med. Jour., 1893.
Hirschberg, Trans. Internat. Med. Congress, 7th sess., Lond. 1881.
Hirschberg, Deut. med. Wochen., 1896.
Hjort, Klin. Monatsbl. f. Augenh., 1867.
Hock, Wien. med. Bl., 1883.
Holtzke, Arch. f. Kinderh., 1894.
Hosch. Cor.-Bl. f. Schweiz. Aerzte, 1883.
Jaeger, Ztschr. f. prakt. Heilk., 1855.
Jessop, Ophth. Review, 1900—Tr. Ophth. Soc. U.K., 1885.
Jessop, St. Bartholomew's Hosp. reports.
Jessop, Ophth. Review, 1883.
Kalt, Ann. de Méd. Scient. et Prat., 1893.
Kirkpatrick, Ann. Jour. of Ophth., 1890.
Knaggs, Brit. Med. Jour., 1892.
Knapp, Ophth. Review, 1892.
Koester, Centralbl. f. med. Wissenschaften, 1874.
Königshöfer, Aerztl. Rundschau, 1896.
Königshöfer, Med. Cor.-Bl. d. Würtemb. Aerztl. Ver., 1896.
Lagrange, Arch. d. Ophth., 1895.
Landgraf, Berl. klin. Wochenschr., 1891.
Lawford, Ophth. Hosp. Rep., Lond., 1888, vol. 12, p. 133.
Leber, Ber. ü. d. Versam. d. Ophth. Gesellsch., 1891, Stuttg. 1892.
Liebrecht, Arch. f. Ophth., 1890.
Lobowski, Arch. f. Augenh., 1897.
Lubowski, Arch. f. Augenh., 1897.
Machek, Przeglad Lekarski, 1892.
Mackenzie, Stephen, Ophth. Rev., 1882.
Manfredi, Ann. di ottal., Milano, 1874, 1875; Arch. d'Ophth., Paris,

Digitized by Google

Manz, Münch. med. Wochen., 1895.
Manz, Arch. f. Ophth., 1858.
Marshall, Tr. Ophth. Soc. U. Kingdom, 1895.
Mazzee, Rev. Méd. de Chile, Sant. de Chile, 1877.
McHardy, Brit. Med. Jour. 1887, 88—Tr. Ophth Soc. U.K., 1888.
Mickel, Sitzungsb. d. phys. med. Gesellschaft zu Würzb., (91) 1892.
von Millingen, Gaz. méd. d'Orient, 1880–81.
Mitvalsky, Bull. et mém. soc. franç. d'ophth., 1896.
Moauro, Atti d. r. Accad. Med. Chir. di Napoli, 1897.
Moore, Internat. Clin. Phila., 1892.
Moraud, Ann. d' Occul., 1844.
Morton, Brit. Med. Jour., 1891.
Motais, Rec. d'Ophth., 1892.
Mules, Tr. Ophth. Soc. U. Kingdom, 1882–83–84.
Mules, Ophth. Review, 1884.
Müller, Inter. klin. Rundschau, 1893.
Nakaisumi, Ztschr. d. med. Gesellsch. zu Tokyo, 1899.
Neese, Arch. Ophth. N. Y., 1887.
Neese, Arch. f. Augenh., Wiesb., 1885.
Norrie, Centralbl. f. Prakt. Augenh., 1895.
Otero y Arce, Gac. Méd. México, 1890, Gac. Méd. México, 1898.
Pagenstecher, La Semaine médicale, 1888.
Palermo, Ann. di ottal., 1892.
Panas, Jour. de Méd. et de Chir., 1888.
Panas et Vassaux, Bull. et. mém. Soc. franç. d'Ophth., 1885.
Parinaud, Gaz. Hebd. de Méd., 1884.
Parinaud, Bull. et Mém. Soc. de Chir. de Paris, 1879.
Parinaud, Ann. d'Oculistique, 1892.
Parisotti, Bull. d. r. Accad. Med. di Roma, 1897–8.
Pasquier, J. d'Ophth, Paris, 1872.
Péchin, Arch. d'Ophth., Paris, 1899.
Perles, Arch. f. Ophth., 1873.
Peters, Zeitschr. f. Augenh., 1900.
Pisenti, Atti e rendic. d. Accad. Med. Chir. di Perugia, 1897.
Pooley, Am. Jour. of Ophth., 1890.
Poucet, Gaz. Med., 1875.
Pregel, Wien. med. Wochenschr., 1893.
Prioleau, Rev. Mem. d. Mal. de l'enf., 1892.
Probsting, Klin. Monatsbl. f. Augenh., 1891.
Pruneda, Rev. Med. de Chile, 1882–1883.
Rampoldi, Ann. di Ottal., 1884, 1888.
Reimar, Klin. Monatsbl. f. Augenh., 1900.
Reissmann, Arch. f. Ophth., 1885, 1888.
von Reuss, Anz. d. k.k. Gesellsch. der Aerzte in Wien, 1887.
von Reuss, Wien. med. Presse, 1888.
Rhein, München. med. Wochen, 1886.
Rochon, Duvigneaud, Arch. d'Ophth., 1898.
Romvolotti, Atti d d. Ass. Med. Lomb., 1894.
Roy, Rev. Clin. d'Ocul., Paris, 1885.
Rüter, Arch. f. Augenh., 1880–81.
Rüter, Knapp und Herschberg's Archiv., 1881–82.
Salzer, Graefe's Arch. f. Ophth., 1896.
Samelsohn, Centralbl. f. Prakt. Augenh., 1879.
Sattler, Trans. Ophth. Soc. U.K., 1897.
Sattler, Irish Hosp. Gaz. Dubl., 1874.
Sandford, Trans. of Oph. Soc. of United Kingdom.
Schapringer, N.-Yorker Med. Presse, 1888.
Schäfer, Klin. Monatsbl. f. Augenh., 1884.
Schell, Tr. Am. Ophth. Soc., 1880–1883.
Schöler, Verhandl. d. Berl. Med. Gesellsch., (1891) 1892.
Schultze, Arch. f. Augenh., 1896.
Secondi, Gior. d. r. Accad. di. Med. di Torino, 1887.
Seeley, Clinic. Cincin., 1877.
Sgrosso, Ist. di clin. ocul. d. r. Univer. di Napoli, 1889–90.
Shears, Liverpool, M. Chir. Jour. 1888.
Silcock, Tr. Ophth. Soc. U. Kingd., 1895.
Silex, Berl. klin. Woch., 1895.
Spicer and Marshall, Tr. Ophth. Soc. U. Kingd., 1897.
Stephenson, Pediatrics, 1896.

Digitized by Google

Stevenson, Pediatrics, 1897.
Stölting, Arch. f. Ophth., 1886.
Stölting, Graefe's Archiv, 1888.
Straub, Weekblad v. Geneesk., 1899.
Story and O'Sullivan, Trans. Royal Acad. of Med. in Ireland, 1899.
Strzeminski, Rec. d'Ophth., 1896.
Stutzer, Beitr. z. Augenh., Hamb. u. Leipsig, 1898.
Swanzy, Ophth. Review, 1882.
Tailor, Ann. di Ottal., Pavia, 1889.
Terson, Arch. d'Ophth., 1893.
Thompson, Trans. of Ophth. Soc. of U. Kingd., 1898–9.
Trombetta, Ann. di Ottal., 1899.
Trousseau, Arch. d'Ophth., 1889.
Truc., N. Montpel. Med., 1834.
Trudeau, Tr. Ass. Am. Physicians, Phila., 1894.
Ulry, Gaz. Hebd. d. sc. Med. de Bordeaux, 1898.
Valude, Ber. ü.d. Versamml. d. Ophth. Gessellsch. Stuttg. 1887–19–66.
Van Duyse, Ann. d'occul., Brux., 1890.
Velhagen, Zehender's klin. Monatsbl. f. Augenheilk., 1894.
Vernon, St. Barth. Hosp. Rep., 1871, 181.
Verrey, Brit. Med. Jour., 1888.
Vicasse, Ophth. Soc. United Kingd., 1897.
Vieusse, Recueil d'Ophth., Jan. 1900.
Villemin, Étude sur la Tuberculose, 1868.
De Lilto Vollaro, Lavori d. Clin. Ocul d. r. Univ. di Napoli, 1897–8.
Wadsworth, Boston Med. and Surg. Jour., 1871.
Wadsworth, Tr. Am. Ophth. Soc., 1880, 1884.
Wagenmann, Graefe's Archiv, 1888.
Wagenmann, Ber. ü. d. Versamml. d. Ophth. Gesell., 1891, Stuttg., 1892.
Wagner, Münch. med. Wochenschr., 1891.
Walb, Klin. Monatsbl. f. Augenh., 1875.
Walb, Klin. Monatsbl. f. Augenh., 1877.
Warner, Tr. Ophth. Soc. U. Kingd., 1882–83.
Warner, The Ophth. Review.
Wells, Trans. Path. Soc. Lond., 1868.
Wells, J. Soelberg, Med. Times and Gaz., 1868.
Wolff, Centralbl. f. prakt. Augenh., 1882.
von Wecker, Wien. klin. Rundschau, 1896.
Weill, Arch. f. Augenh., 1893.
Weinbaum, Archiv. f. Augenh., 1894.
Weiss, Arch. f. Ophth., 1877.
Winter, Ztschr. f. Fleisch- u. Milcher-hyg., 1898.
Wolfe, Brit. Med. Jour., 1882.
Wordsworth, Med. Times and Gaz., 1860.
Zieminski, Rec. d'Ophth., 1886–88.
Zimmermann, Medicine, Detroit, 1897.
Zimmermann, Rec. d'Ophth., 1898.
Zimmermann, Rec. d'Ophth., 1888.

DEVITALISED AIR TOXÆMIA—A PRIME CAUSE OF TUBERCULOSIS.

By CHARLES DENISON, A.M., M.D. (*Denver, Colorado*).

To show the importance of recognising the *basic law of degeneracy* which springs from rebreathing once-used air, and which finds its chief and final expression in that wasting disease, tuberculosis, is the task I have set myself to perform.

It is incumbent upon us to appreciate that there is a deeper and more fundamental source of knowledge than that which the microscope, with its wonderful delineation and detail, can supply, and that the liability of being side-tracked by that instrument's findings should be recognised in order that we may not fail to grasp the true origin and evolution of this degenerative disease.

Digitized by Google

The term *devitalised* is new only as applied to the air which has been breathed. Sir B. W. Richardson, in his work "On Certain of the Phenomena of Life," used this word to designate the quality of oxygen in which animals died after respiring part of it, they being unable longer to live, though only one-eighth of the oxygen had been consumed. To quote from the "Smithsonian Contributions to Knowledge" (Report by Drs. J. S. Billings, Weir Mitchell, and D. H. Bergey, vol. xxix., 1895):—

"Richardson, in 1860–61, found that a temperature much higher or lower than 20° C. had the effect of shortening very considerably the lives of animals confined in an unventilated jar, and that these effects were more marked when the animals were confined in an atmosphere richer than air in oxygen. In this case he found that by passing electrical sparks from a frictional machine through the *fatal* air (previously deprived of carbonic acid) he made it again capable of supporting life; from which he concluded that oxygen is *devitalised* during respiration, and that the electric spark has the faculty of revitalising it."

By the term *toxæmia* is not here meant the sudden asphyxiation from carbonic acid poisoning, nor the rapid starvation from lack of oxygen (one or the other of which had to do with all the experiments carried on under the direction of the Smithsonian Commission above referred to). Our definition refers to that lesser vitiation of the blood and living tissue (better, perhaps, called a *dyscrasia*) which gradually, through accumulation, results in degeneration, which is the basis, or so-called "soil," of disease, rather than any at present recognised distinct affection.

The life of the air consists, to a greater extent than has been heretofore recognised, in the molecular mobility of its atoms, caused by the sun's influence. The diffusibility of the air, its easy and ceaseless motions, due to changes of temperature in different strata, are forms of molecular activity which, under the influence of some electrical or other force yet to be fully understood, probably impart the life-giving principle to the atmosphere.

The restraint of this molecular motion and the consequent limitation of vitality are in direct proportion to the deficiency of ventilation. Herein I conceive lies the great mistake of our civilisation, *i.e.*, in relation to our mode of living. Here is to be seen the need of education that this cause of disease may be realised.

As to the Richardson experiment, I believe we must have further laboratory proof of this kind before the true character of devitalised air toxæmia shall be understood and generally accepted.

If such distinguished physicians as Billings, Weir Mitchell, and Bergey failed to recognise the importance of this inquiry, it is not surprising that we, too, have not grasped its full meaning.

The Report of these men upon "The Composition of Expired Air and its Effect upon Animal Life" was recommended to the Smithsonian Institute for Governmental publication by a committee of distinguished physicians (Dr. H. C. Wood and Professors W. H. Welsh and C. S. Minot). Yet, having acknowledged in the body of their work (as quoted) Dr. Richardson's important discovery, and having repeated his experiment, *imperfectly* in that no revitalisation of the air was attempted by static electricity, they ignored his discovery

Digitized by Google

in their conclusions. Why did they attempt Dr. Richardson's experiment and not make it complete? They went all round it, and showed by inference the need of such a determination.

This is shown by the following quotation from the Report in question :—

"It is, however, by no means demonstrated that the only deleterious effect which the air of crowded barracks or tenement-house rooms, or of foul courts and narrow streets, exerts upon the persons who breathe it, is due to the greater number of pathogenic micro-organisms in such localities. It is quite possible that such impure atmospheres may affect the vitality and the bactericidal power of the cells and the fluids of the upper air passages with which they come in contact, and may thus predispose to infections, the potential causes of which are almost everywhere present, and especially in the upper air passages and in the alimentary canal of even the healthiest persons; but of this we have, as yet, no scientific evidence. *It is very desirable that researches be made on this point.*" (Italics mine.—C. D.)

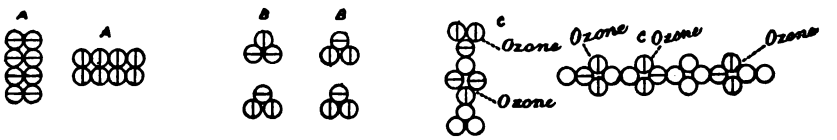
Their own "Richardson experiment" showed that there was enough excess of oxygen left (40 to 61 per cent.) in the sealed jars containing the dying or dead animals to support life, and they had previously shown that death does not necessarily occur from the excess of carbonic acid remaining. Richardson asserted that death occurred, though not so soon, with that carbonic acid removed from the oxygen which had been breathed but only partly consumed.

The logical inference is that Richardson's original experiment stands *not yet disproven*. Furthermore, it is, by inference at least, greatly strengthened by Paul Bert's experiments on "Atmospheres containing an excess of Oxygen,"* as well as by Dr. Bergey's experiments quoted by this Commission.

When needed additional experiments shall have been made and Richardson's finding generally recognised and taught as a fundamental principle, we shall have made this important advance in knowledge, namely, the certainty that some as yet unknown condition in the composition of the air, or in the arrangement of its ultimate atoms or molecules, is essential for the sustenance of life, and that breathing changes that condition or arrangement, *i.e.*, *deprives it of vitalising power.*

It is said that the atoms of the oxygen molecule go in pairs, *i.e.*, oxygen has two atoms, ozone three. Let me suggest, by way of illustration, that perhaps, without the agency of atmospheric electricity (as in used or breathed air), the poles of these pairs are always parallel; but with that agency operative the needed disturbance or correction occurs which will throw some of the poles of these oxygen atoms at right angles to others, and thus, to a certain extent, three atoms of oxygen will come together, making ozone, which perhaps may be the life-giving principle in the air.

To make this plainer, let A A represent the possible arrangement of the oxygen atoms in pairs; B B that of ozone. Then the following arrangement, C C could be assumed to be the possible breaking up by static electricity of the oxygen arrangement to make ozone :—



* "Leçons sur la physiologie de la respiration." Paris, 1870, p. 510.

Digitized by Google

This is put forward only as an hypothesis in order possibly to open the way for some chemist to make plainer this obscure action of the atmosphere. Even when explained, should this arrangement—presumably due to atmospheric electricity—be found to be correct, it would need to be definitely determined how much of the vivifying influence caused by electricity, in once used and unventilated air is due to the purifying, disinfectant ozone influence upon exhaled poisons.

There may be a new principle or ingredient yet to be discovered in the air, which governs this arrangement either of its atoms or molecules, which thus makes it respirable. Indeed, we seem to be confronted with a most important inquiry, the answer to which would be of inestimable value, namely, What is the relation of atmospheric electricity to the respirability of the air? In other words, how does breathing the air, though its oxygen be only partly consumed, make it unfit for again sustaining life till it is re-electrified or revitalised? Or, again, what, if any, is the inhibitive state of the oxygen in the air, especially in once-used air, which renders it non-absorbable or its use nugatory in sustaining life?

As to bacteria, and their ability to change the chemical qualities of the substance upon which they live, thus causing the degeneration of lifeless forms of existence to lower states, we do not know that oxidation retards that decadence. Hueppe's book abounds in fine illustrations of these effects.

NOTE.—"The Principles of Bacteriology." By Dr. Ferdinand Hueppe, Prof. Hygiene in the University of Prague. The Open Court Publishing Company, Chicago.

It is for a biologist like Prof. Ferdinand Hueppe to take up this subject and elucidate it, for he reasons that, "in principle at least, the biologist is at no disadvantage in the matter of accurate prediction as compared with the physicist or astronomer."

NOTE.—Prof. Hueppe, in discussing the action of electricity on bacteria, says, "The electric current acts upon toxic bacterial cultures in such a way as to remove their toxicity, and cultures treated in this way, with their toxic power destroyed, are able to confer immunity."

To make a few quotations (p. 93), he says:—

"It is possible to pass from inert, lifeless proteid to the active living proteid, and Scholl succeeded in one case in making again active a proteid which had been rendered passive through heat."

"It now appears clear from chemical considerations that in anaerobiosis, oxygen, to speak exactly, is not taken out of the molecule. There is simply an atomic re-arrangement by means of which the hydroxyl radical (O H) affects the oxidation of the carbon to carbon dioxide, and either hydrogen is liberated, or reduction-products rich in hydrogen arise, or hydrogen combines with sulphur to form the sulphuretted hydrogen. The instability of the atom grouping is therefore the real primary reason for the breaking up, and this process is in certain cases independent of oxygen, and can therefore proceed anaerobiotically.

"Cholera cultures are virulent when young, first becoming impotent *through access of air*. For the same reason our cultures of pathogenic bacteria upon gelatin agar and potato, and in broth, are generally without power to produce disease, since, in consequence of the restriction of metabolic activity to the splitting of certain substances, they have accustomed themselves to a metabolism based on oxidation, that the poisons that were formed in the first instance through decomposition are rendered harmless through the subsequent oxidation. Ultimately, therefore, the formations of these poisons comes to a standstill."

The toxæmia we are considering seems to be due to stagnant unventilated air existing in the lungs themselves. It is not necessary, in this audience, to

Digitized by Google

show that ventilation, though ample outside the body, may be very deficient within the lungs, because of disuse from insufficient exercise. The force of our argument increases as we go inward with the analysis of the air. The vitiated and fermenting combination of the unrenewed pulmonary air, increased by body heat, the outcoming carbonic acid and the poisonous tissue detritus, is simply a tenfold intensification of devitalised air outside that body in an unventilated room. The living tissue that will stand this and not deteriorate must indeed be in prime condition.

This devitalisation, due to lack of ventilation, leads to the pale face, sallow skin, weak pulse, cold hands and feet, and sluggish bowels; the feeble powers of digestion, assimilation, and nerve energy—all proofs of flagging vitality. This lethargy is due to enfeebled and poisoned blood-corpuscles, and a probable auto-infection, but especially to deprivation of food for the blood, *i.e.*, oxygen from the air. In addition, the *dead air*, which marked deficiency of ventilation implies, compels inactivity and limited use of the lungs, and this in turn means the gradual clogging of out-of-the-way air-cells with the products of combustion. In this manner, carbonic-acid poisoning and a species of auto-infection are more or less permanently established.

The destructive influence of sunlight upon the tubercle bacillus is now generally admitted. If, in addition, the contention of Frankel—that this bacillus is a facultative anaerobic germ—is sustained, then the claim of a predisposing cause in defective ventilation is strengthened by the existence of favourable conditions furnished for such germination in the warm, unventilated lung.

Why cannot the possibility of this dyscrasic state and its cause be understood, and then generally accepted as the basis of prevention and of proper administrative control? Let us examine the prevailing belief as to the cause of tuberculosis and find an answer.

Since the discovery of the bacillus of tubercle, a dangerous circuit of thought has become prevalent among medical men with reference to consumption. I say dangerous, because it narrows the field and limits the scope of that education which we all admit is essential to the successful issue of the present tuberculosis crusade. I object to this tendency because it leads to a circuitous and superficial way of thinking, and is a hindrance to right conclusions. Three quotations will suffice to illustrate:—

(1) Dr. Edward O. Shakespeare (in his paper read before the Association of American Physicians in 1890) voiced the *dictum* of the learned discoverer of the bacillus of tubercle when he declared: "There is no fact within the whole range of medical knowledge for the demonstration of which a greater mass of the most exact, positive, convincing proof can be marshalled than that the bacillus tuberculosis is the sole, active, exciting cause of tuberculosis, and that without the agency of the micro-vegetable parasite there can be no genuine tuberculosis."

(2) More recently the thought was tersely put as follows editorially in the *Journal A. M. A.* (p. 1413, Dec. 1st, 1900): "There can be no tuberculosis without the bacillus, and the disease is almost exclusively transmissible

Digitized by Google

through matter containing the micro-organisms, and, as the lungs are so generally involved, most commonly through the expectoration. The proper care of the expectorated matter, therefore, would alone go a great way in reducing the morbidity to and mortality from tuberculosis."

A similar trend of reasoning is characteristic of Prof. H. M. Biggs' fine address on "The Registration of Tuberculosis" before the Philadelphia Medical Society, Nov., 1900, and of an excellent statistical article read before the Michigan State Medical Society last July (see *Philadelphia Medical Journal* for Jan. 12, 1901) by Dr. Collins H. Johnson. To summarise: The great, almost universal, prevalence of active or latent tuberculosis in adults being instanced, as shown by post-mortem evidence of tubercular lesions found in over 4000 autopsies in Breslau of 33⅓ per cent.; by Biggs in New York, 60 per cent.; by Brouardel in Paris, 75 per cent.; and by Otto Naegeli, in the Pathological Institute of Zurich, from less than 30 to 99 per cent., according to the age; therefore the infectious character of the disease is paramount. This germ-limited vision of the subject pervades scores of medical papers written all over the world which, like the rehearsal of a creed, announce belief in the germ—the existence of which nobody questions—and starting with it, work out from it and back to it, so that etiologically the germ is the beginning and end of it all.

Following such authority everybody seems compelled to look at this disease through the coloured glasses of a dogmatic bacteriology, which fails to recognise that long formation period when stunted growth and false building of tissue are characteristic of the affection. Thus the pre-tubercular stage is slighted, however admittedly explainable are the infiltration, softening, and excavation stages on the germ theory. No bacillus, no tuberculosis; all consumption as well as scrofula is, of course, tubercular, therefore, none of these without the bacillus—*ergo*, the destruction of the bacillus is demanded. Since the bacillus is found chiefly in the sputum, laws against or governing expectoration are to be placarded everywhere, and the reason is therefore presumably warranted for the registration of all tubercular persons, for the instruction of State and other boards of health!

We must not alarm the people. Rather, they must be "educated" against this terrible bacillus; how to destroy it when it has been born and grown up, and how to clean up after their friends or relatives have died from its ravages.

Hueppe says, "Education to a state of panic is not a wholly new discovery, and the earliest products of the scare system are certainly just as ridiculous and disgraceful as the newest."

As to the quotations from Dr. Shakespeare and the medical journals:

(1) It never has been and never can be shown that the *bacillus* is the "*sole*" cause of all the conditions classed as tubercular, for *other* causes which produce *susceptibility* are entitled to quite as much consideration.

(2) It is wholly presumptive to say because "the lungs are so generally involved that the disease is almost exclusively transmissible through the expectoration." The function of the bronchial glands, the part played by the

Digitized by Google

delicate, unventilated lung tissues, and the retention of *other* poisons than the supposedly inhaled bacilli—all these facts need to be considered.

(3) The statistics of Biggs and Johnson cited a large portion of humanity as having been at some time or in some degree infected with tuberculosis, and that, too, increasingly with the advanced age of the individual—an increase which, taken with the evidence of H. Schreiber that new-born infants do not react to the tuberculin test, shows that the human race probably starts out free from the disease. This ought to prove, or at least to suggest, that there must have been a condition precedent for this disease acquired during life from peculiar environment. Such a fault of our civilisation, if we can only find and acknowledge it, will explain the frequent occurrence of this disease. Then, to the reflective mind, this most fatal scourge of the human race is as clearly due to *nonconformity to healthful environment* as, according to our belief, was the extinction of the mammoth megatheriums and kindred animals due to geological changes in their environment.

A year ago I endeavoured to show before the American Climatological Association (*see* paper on "The Degenerative Results of Deficient Ventilation," read before A. C. A., 1900) that this fault of our civilisation is chiefly due to the bad construction of our houses, in that their allowance of air space and motion, sun-warmth and atmospheric electricity, are curtailed until they become insufficient for healthful life.

Let us illustrate this thought by the comparison of curved lines (see chart) representing the average duration of life under (1) normal conditions as to perfect ventilation; (2) the shortening of life by devitalised air toxæmia through deficient ventilation; and (3) another line representing Dr. Angus Smith's extinguishing candle-light curve. The records of this indefatigable investigator show that the candle in his closed lead chamber grew dim in proportion as the curve is drawn in the accompanying diagram, and was extinguished when the amount of carbonic acid generated reached 2 per cent. We note in passing that Dr. Angus Smith could remain in his tightly closed chamber after the light had gone out. Further, in contrast with the light's curve, we remark that tuberculosis free or normal life curves run nearly horizontal during the first two-thirds or three-fourths of the average duration of life, and then, in old age, drop off suddenly to the death line.

Now as to the deficient ventilation curve. The life of the air exists chiefly in its motion, which, with its light, heat and electric power, comes from the sun. If the absence of this life, here designated as *dead air*, is represented as a perpendicular line, and, in contradistinction, *live air* is represented as another straight line at right angles thereto, then the gradations from live air to dead, under the subtle influence in which we human beings live and die, can be truthfully represented as a curved line which gradually leaves the horizontal line and comes down to the perpendicular. While this drawing of the deficient ventilation curve is admitted to be only approximate, it is not entirely arbitrary; for an effort has been made to have it correspond to the average deficiency of ventilation under which the inhabitants of a city

Digitized by Google

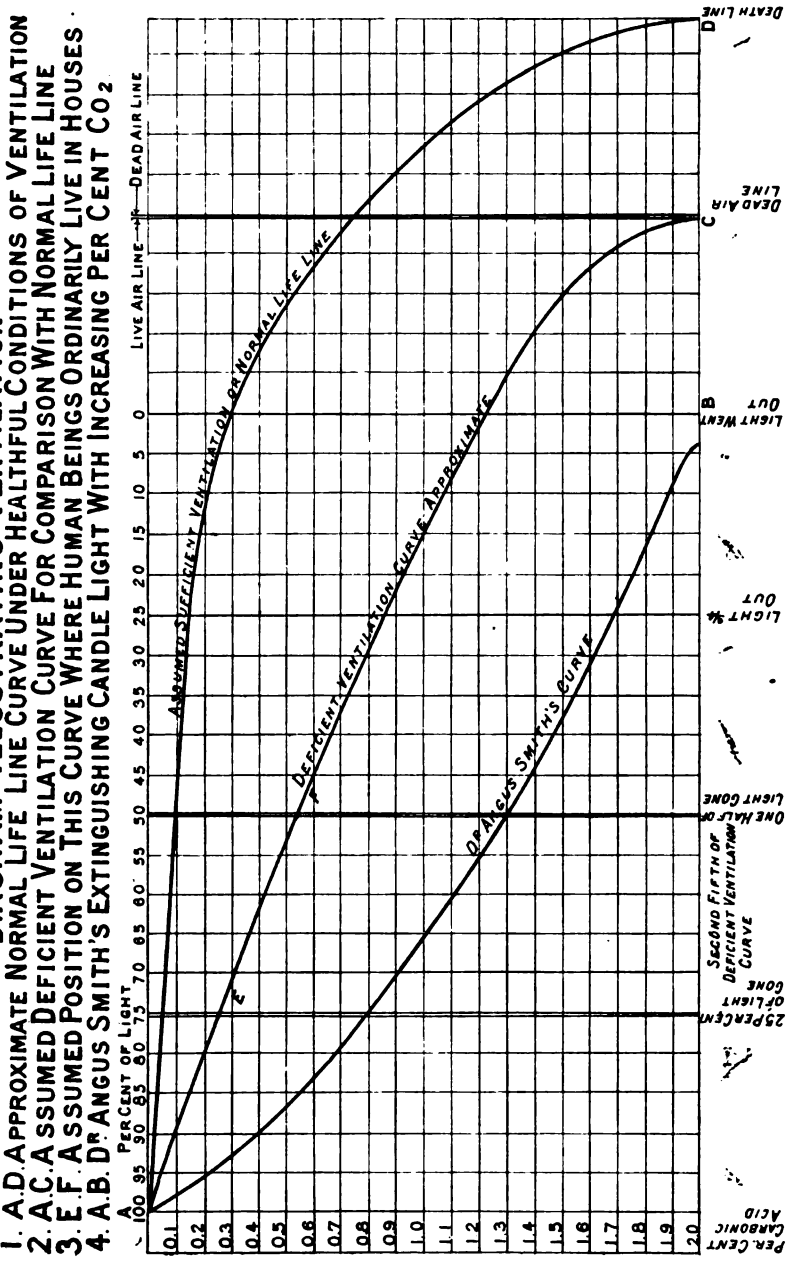


DIAGRAM ILLUSTRATING VENTILATION
1. A.D. APPROXIMATE NORMAL LIFE LINE CURVE UNDER HEALTHFUL CONDITIONS OF VENTILATION
2. A.C. ASSUMED DEFICIENT VENTILATION CURVE FOR COMPARISON WITH NORMAL LIFE LINE
3. E.F. ASSUMED POSITION ON THIS CURVE WHERE HUMAN BEINGS ORDINARILY LIVE IN HOUSES
4. A.B. DR. ANGUS SMITH'S EXTINGUISHING CANDLE LIGHT WITH INCREASING PER CENT CO_2
PER CENT OF LIGHT
LIVE AIR LINE
DEAD AIR LINE
ASSUMED SUFFICIENT VENTILATION OR NORMAL LIFE LINE
DEFICIENT VENTILATION CURVE APPROXIMATE
DR ANGUS SMITH'S CURVE
PER CENT CARBONIC ACID
25 PER CENT OF LIGHT GONE
SECOND FIFTH OF DEFICIENT VENTILATION CURVE
ONE HALF OF LIGHT GONE
LIGHT ¾ OUT
LIGHT WENT OUT
DEAD AIR LINE
DEATH LINE

Digitized by Google

of 100,000 persons live, taking as a basis the approximate cubic space *per capita* in their sleeping and living rooms. On this deficient ventilation curve I calculate that the unhealthful conditions on the other side of the Atlantic, and I presume on this side also, place the average of people (see diagram, E to F) 20 to 40 per cent. along on its descent toward the death line.

We do not appreciate our individual positions on this ventilation curve, as to the respective confinements of our bodies in closed spaces and the resultant imprisonment and dwarfing of our respiratory and circulatory systems. It then becomes a pertinent question to each one of us, Shall we be exterminated by these conditions, or will the final extinguishing act be transmitted to our children, with their impaired vitality caused by our present ignorance?

Let us now consider the subject of susceptibility and immunity to tuberculosis from a *pre-germ* standpoint. With circulation, respiration and nervous power perfect—the defensive body cells (the leucocytes and phagocytes) being abundant and on the alert—there is very little probability, even if there is a remote possibility, of an ordinary amount of tubercle bacilli infection finding a permanent lodging in the human body.

NOTE.—Hueppe attributes to H. Buchner the first demonstration of the remarkable fact "that blood serum and active tissue juices, fresh milk, etc., possess the ability to kill bacteria, and sometimes display extraordinary activity in this direction." Behring next recognised that active blood serum can nullify the effect of bacterial poisons.

Knopf (*Journal A. M. A.*, June 15, 1901) says: "The healthy nasal secretion is bactericidal; the ciliated epithelium of the upper respiratory tract is a physical hindrance to the bacilli. The gastric secretions, too, are in a large measure bactericidal."

Such a resistance of the body makes it to this extent immune, but every organism has its immune limit to such an infection as tuberculosis.

Immunity to tuberculosis, then, is always relative. It is so in two ways: (1) relative to the strength and health, *i.e.*, the resistant state of the individual; (2) relative to the amount and quality of the infecting material. The protecting skin of the body and normal linings to reachable cavities are great aids to immunity and opponents to susceptibility. So in perfect health, whether in infancy or in age, we are practically immune and insusceptible to tuberculosis. This is an important point to admit. It is the condition in Nature we ought to enjoy.

This is the state of practical freedom from tuberculosis possessed by all wild animals living out in the open—a state of freedom once enjoyed by the wild Indians, now, however, unknown to them when civilised; an immunity even now retained by cattle living in a *natural* wild state on the western plains, but lost by the same cattle when packed in four by seven foot stalls in practically (but not actually) underground stables; in even worse condition as to ventilation than is a crowded modern sleeping-car.

Of all the varying phases of human existence which break down immunity and ring in susceptibility, that here cited, I believe, is most fruitful of harm.

As man advances in civilisation he becomes more confined and less active, and therefore needs to give more thought to his environment in order to assure physical health. The higher the order of evolution (*i.e.*, the more

Digitized by Google

delicate and special the development of the nervous structure), the more susceptible to and influenced by the changes in environment are all created beings.

Applying this principle to the human family, we see that a limit of civilisation can be reached where delicate or slight changes of environment may cause dissolution as well as evolution; where extinction from *lack* of conformity may take the place of evolution, *because* of conformity to our environment. We believe that the sun is the source of all life, and hence is the means of sustaining healthful activity through the medium of the air. Then, aside from that contribution of the sun's store-house classed as food, it is evident that the air we breathe carries in its oxygen, temperature and electrical state the life we need, and presumably enough of it, taken *as it was intended to be used.* That any vitiation of the forces of that source of life, leading to any fault or disease of body, will be chiefly manifest in the breathing organs goes without saying, and must be admitted to be a reasonable explanation why tuberculosis generally starts in the lungs.

With advancing civilisation (the development of a man greater physically and more active mentally) an increased amount of air *per capita* is demanded. There is no lessening of the amount possible nor of the invigorating quality required. On the contrary, under certain conditions of life—like altitude, because the air is thinner, and in certain workshops because the air is specially impure—proportionately more is required. How much is needed? This is the greatest question of the hour. We do not know very definitely. We speculate, and then, because of our ignorance, we compromise on the side of economy. The result is gradual decay, tuberculosis, and death.

Studying this matter I assumed as necessary a minimum of 2000 cubic feet *per capita* renewed once per hour; and even on that basis (which is probably too small for perfect safety) decided the human race is probably living on a deficient ventilation curve. It is literally starving its breathing and blood-making organs. Physicians interrogated estimated that people in their various cities would not average half that, some even guessing as low as 500 cubic feet *per capita*, and you know that any *change per hour* is the exception and not the rule.

Considering the evidences of the general freedom from tuberculosis under favourable conditions, and on the other hand referring to the general prevalence of the disease under conditions worse as to ventilation than that above stated, and also to the blood changes induced by breathing such devitalised air, I want to ask, Is it reasonable to substitute the germ results for such causes when the latter are so plain? Do not the changes in the clogged-up vessels and air cells, induced by thus rebreathing devitalised air under conditions of physical restraint, possibly constitute a *culture field* which could not exist in a normal lung? Beyond question this condition could be so bad without germ infection that it could not be much worse with it, so near to a created toxæmia is the dyscrasia existing.

NOTE.—Hueppe (p. 114) concludes: "Bacteria, then, are able to construct their body substances out of various kinds of nutrient materials, and also to produce the organic pigments or fermentation products or poisons especially characteristic of individual species,

Digitized by Google

and they are able to do this either analytically or synthetically with almost equal ease. This ambidextrous metabolic power exists among bacteria to an extent unknown as yet among any other living things, and these organisms consequently occupy physiologically even more than morphologically a kind of *intermediate place between animals and plants.*"

Do we not seem to be just on the border of what Prof. Hueppe refers to in declaring, "Tuberculosis occurs spontaneously in apes as well as in man and in cattle"?

NOTE.—Prof. Edward O. Jordan, who translated this work, writes as follows in regard to this quotation :—

"I suppose Prof. Hueppe would explain the statement you refer to as having reference to the natural contracting of the disease in contradistinction to the production of the disease by inoculation. That is to say, guinea-pigs and rabbits, although susceptible to inoculation with tubercle bacillus, do not contract the disease spontaneously under natural conditions. That, I take it, is the author's meaning."

On the contrary, Hueppe himself seems to be ready to accept spontaneous generation as explaining the origination of these germs, for he thus concludes his chapter on "The Structure of Bacteria": "After all is said, spontaneous generation remains up to the present time only an unavoidable general hypothesis." He further states that "indirectly these results (bacteriological researches into the synthesis of proteid substances) perhaps support the theoretical postulate of spontaneous generation, since they seem to efface still further the boundaries between animal and plant, the organic and the inorganic."

I do not wish to introduce the diversion of a discussion of the spontaneous generation of life in these mould growths, which these bacilli of tubercle are acknowledged to be, for I am not aware of any experiments which conclusively prove this. But this I do say, that if, in warm-blooded animals, marked deficiency of ventilation, prolonged even perhaps for generations, does eventually in whole or in part produce such a biological metamorphosis, such a change *in* the blood and tissues of their breathing organs, as to result in the formation of this vegetable mould-growth, then such spontaneous generation, to my mind, is simply another way of saying that this dyscrasic state is a necessary precursor to a germ infection from *without* such animal body. The dyscrasia is necessary in either case. Either supposition will help us to get at the *real* cause of tuberculosis, *i.e.*, that which precedes the germ formation. Here, indeed, is susceptibility in the extreme, and immunity is correspondingly lost. Up to a certain point germs have yet nothing to do with it, but a condition already exists possibly as dangerous and deadly in one person as a plentiful incubation of myriads of bacilli in another.

Here is a toxic condition before any positive evidence is visible of germ existence, when the tuberculin test will give distinct reactions about the same as when the bacilli are known to be present. What is it? Isn't it the same disease that it will be, if allowed to go on to degeneration and disintegration of the affected lung tissue? Supposing, at this point, before known germ formation, the patient has recovered. From what has he recovered? Is it a jump from the *pre-tubercular* to the *post-tubercular* stage, without any active tuberculosis ever having existed as discoverable by germs? Were or were not both of these *pre* and *post* conditions included in the Breslau autopsies, in Biggs', Brouardel's and Naegeli's post-mortem results? If so, are we not driven to the conclusion that the term tuberculosis should cover the whole process? If not, we must invent a new name for this toxæmia. When germification commences, and how it commences, will be interesting to learn, for our suspicions grow apace as the vegetable-mould nature of the germ is demonstrated.

NOTE.—P. 143, Hueppe says: "The so-called tubercle bacillus is not a bacillus at all from the point of view of bacterial classification, but a parasitic growth-form of pleomorphic mould." P. 144: "The ability of bacteria to form pigments, fermentation products, and poisons, and to provoke disease, is proved by accurate investigation to be simply a quality of adaptation."

Digitized by Google

However, *the cause*, due to defective aeration (breathing devitalised air), preceded the germ in any event, and we are not going to profit anything by denying this fact. On the contrary, we have everything to gain by recognising it; for here are both the prevention and the cure brought within our easy grasp.

Let me propose to you, gentlemen, an hypothetical question.

What would be the difference in two classes of people at the end of the first, second and third generations, as to mortality from tuberculosis?

The two classes are to typify lives in separate atmospheres, represented by, for the first class, short measure, still and sunless air; for the second class, sufficient and stirring air, with sunshine.

Supposing a thousand persons equally divided as to sex and age (16 to 30 years) were to constitute each class.

Each class is supposedly composed of equally hard workers in some factory employment, without vacation or intervening recreations.

Choose for them a good healthy climate, as good as or better than the average of the whole United States. The two classes are to start exactly alike, perfectly healthy, with no tuberculous taint, to be supplied with plenty of wholesome food, with no outside causes of infection, as through tuberculous milk; and no defective drainage or any contagious diseases to be allowed to complicate the test. Each class to reside by itself and to intermarry among its own class; the only difference to be in the quality and supply of the air they breathe. First class to have not over 800 cubic feet of air *per capita*, either in sleeping, living or factory rooms, all occupied without ventilation or direct sunshine. Second class to have 3000 or more cubic feet per person, changed at least once per hour, and to have direct sunlight in all living, sleeping and factory rooms.

Your calculation of the difference in susceptibility and immunity of these two classes should be your estimate of perfect ventilation as a prevention of tuberculosis. Physicians in busy professional life, especially those of this Congress, being representatives of large cities, can determine from their individual experience that this is not an hypothetical question impossible to answer, though the reply must necessarily be expressed in general terms. You can fairly picture to yourselves the divergence, increasing with time, between the growing susceptibility to tuberculosis of the first class and the more stable immunity of the second?

As to my own answer to such an hypothetical proposition, I do not believe that all the skill and knowledge that God has given to man, applied through foods, drinks and drugs (toxins and "anti-toxins" included), to stem the tide of degeneration in that first class, could prevent it—at least, in the third to the fifth generation—from becoming a hot-bed of tuberculosis.

In the vegetable world, Nature eventually supplies the *soil* with its proper and congenial *seed*. This new life found in active tuberculosis, though vegetable in character, only grows in opposition to our life because the soil in us has degenerated to a state fit for its fructification. The *soil* in man is only what he has made it. It is decaying and rendered suitable for germification

Digitized by Google

because the dead-air breathed, the air killed as to its life-giving oxygen and electricity, has transferred its condition to the live animal tissues involved and made them fit only for a lower form of existence.

NOTE.—Hueppe (p. 141), in speaking of the tubercle bacillus, says: "It is just this belief that has made the science of bacteriology so popular in the eyes of the unreflecting multitude and of many easy-going physicians. A fatal blow is dealt to these self-exceptions and illusions by simply pointing to the fact that bacteria provoke fermentation only when they come in contact with fermentable substances under proper conditions, and produce illness and disease only when predisposition to disease exists!"

"Koch's belief that the cause of disease is the constancy of the 'specific' disease-producing bacteria renounces at the outset a scientific explanation. With the abandonment of Koch's position, which has been made untenable by the facts discovered in the past ten years, bacteriology has progressed beyond the stage of a natural history subject, and advanced to a truly scientific standpoint."

And what is the meaning of all this?

That tuberculosis mainly springs from, and must be the outgrowth of, a common predisposing or uniform cause which will account for its finding a congenial "soil" in birds, cattle, horses, and other animals, as well as in man.

The conclusion is almost forced upon us that the medical profession has been trying to proceed with "the cart before the horse"; that it has assumed an *accompaniment* to be a *prime cause*, and has put it in the lead, when it was only a secondary issue, a trailing cart which should come behind. Of course, then, on this basis, we are all wrong in our methods of administrative control.

We have had our hobbies and experiments, so did those who thought to get a desirable *equable* temperature down in the mammoth cave of Kentucky and came near ending the days of all the sixteen consumptives they took down there to be cured. Let us get back *of* the germ and its deadly environment to the prime cause of tuberculosis.

The first thing to do is to set on foot the most thorough and searching investigation possible to determine the *per capita* need of air and ventilation, in order that the predisposition to tuberculosis may be prevented.

Would that we had an automatic deficient ventilation indicator, which when built into the side of a room would announce by sound or a visible index any degree of deficiency. Such an invention, set at a liberal gauge for purity and freshness of air, and universally used in living and sleeping rooms, would help to banish tuberculosis from the earth.

It goes without saying that the second thing to do, having determined what is sufficient ventilation, is to work through educational and administrative channels to have proper laws put in force to remedy the evils of improper house building and overcrowding characteristic of our age. Necessarily similar laws must apply to the housing and hygiene of milch cows, so liable are they to infect human beings, especially children.

NOTE.—Then when the above has been accomplished, a properly officered and supported Board of Health will accomplish something more than it has hitherto. To such a Board will be submitted the plans of all buildings, both public and private, and after construction it will have supervision of their ventilation and over-crowding. Then it will not seem so

Digitized by Google

necessary to register cases of tuberculosis, for, on complaint of a fellow-workman or any one, as in an over-crowded "sweat shop," of a case of tuberculosis forced into close companionship with others, the medical inspector will investigate not only the physical condition of the person complained of, but his *whole environment.* Then it will be possible for the Board to report such actively tubercular person for *banishment from the city to the country*, or to where healthful conditions and non-contamination of others will be assured. The Board of Health should have full power to remedy the evil, which will be done without resorting to any farcical compulsory registration.

Much valuable knowledge that now seems to be Utopian would result by the adoption of such preventive measures. The law, the administrative control, is then based upon prevention, and prevention rests upon a knowledge of the *prime cause* of the disease. The State can then say to the individual, *build your houses thus, and so keep them ventilated, otherwise live in the open air.*

The conclusion is that we, the human race, are living *in nonconformity with our proper environment*, the correction of which fault would be comparatively simple had we the needed intelligence.

THE CHAIRMAN (DR. C. THEODORE WILLIAMS): We are very gratified by the excellent communication of Dr. Denison. Dr. Denison is a well known authority on climatic and other subjects in America, and the great upshot of this interesting communication is to urge ventilation, and I must say I think, looking back on the excitement of the last two or three years, we have been looking too much in the case of tuberculosis, or if not too much perhaps a great deal to the question of sputum, and we have not thoroughly investigated one of the most important causes—it may be pre-disposing causes —*viz.*, ventilation. My impression is that in this great city a large amount of tuberculosis is really caused by bad ventilation; but if, as Dr. Denison has pointed out, everybody had 2000 cubic feet of air, which was changed every hour, I think you would find that tuberculosis would disappear like magic, but that, of course, is almost impossible. The conditions under which we live make it very difficult, but still that is the standard we ought to aim at. I have had the pleasure of visiting Dr. Denison's home, and I therefore know that he carries out what he thinks is necessary, and perhaps that is the reason why he looks so well. But we must never forget, as regards ventilation, the men who taught us so much—Dr. Guy and Mr. Southwood Smith. They taught these doctrines fifty years ago, and they certainly got certain Acts of Parliament passed which have been of great advantage. All we have to do is to add to those Acts and see that they are properly carried out. We are much indebted to Dr. Denison for his paper.

Digitized by Google

FREQUENCY AND SIGNIFICANCE OF TUBERCULOSIS OF THE TRACHEAL AND BRONCHIAL GLANDS IN MILIARY TUBERCULOSIS OF CHILDREN, AND IN TUBERCULOUS MENINGITIS.

By P. HAUSHALTER, *Agrégé at the Faculty of Nancy;*

AND

A. FRUHINSHOLZ, *Hospital Interne.*

In 78 autopsies of miliary tuberculosis in children below 12 years of age, tuberculosis of the tracheal and bronchial lymphatic glands was met with 74 times (about 95 per cent.); in 67 cases there was tubercular meningitis, and in 64 of these affection of the glands was present.

This glandular affection has always the character of an old and caseous tuberculosis; in 29 cases it coincided with old tuberculous lesions of the lung, generally localised; in 44 cases it formed the only old tuberculous lesion present.

In almost all cases, therefore, the origin of the bacillary infection which ends in the miliary form and in meningitis, is in caseous tracheal and bronchial glands. The frequency of this lesion is a proof of the importance of aërial contagion of tuberculosis in children.

How does this tuberculous affection of glands, which is more developed and deeper-seated in the miliary than in any other form of tuberculosis, end in the bacillary infection of the blood, which is considered almost the sole cause of the miliary form and of the meningitis? In spite of the intimate relations of the vessels at the base of the heart with the masses of caseous mediastinal glands—relations which are shown by the drawings exhibited—we have failed to find, macroscopically or microscopically, any lesion of the walls of the vessels that could explain the direct passage of the bacilli from the lymphatic glands into the blood.

The bacillary infection of the blood takes place though the lymphatic system; in the very brief time occupied by the passage of the bacillus-bearing leucocytes from the caseous lymphatic-glands of the mediastinum into the blood stream, passing as they do by the efferent lymphatics, and by a short section of the thoracic duct or of the great lymphatic trunks—the destruction of the bacilli has not time to be effected.

Even in many of the cases in which pure meningitis exists, without generalised miliary infection, and in which one would therefore be tempted to look for the origin of the infection, not in the blood-vessels but in the lymphatics, it is probable that there exists a general bacillary infection, the manifestations of which are not appreciable to the naked eye, or are exceedingly scattered. This we have been able to prove on several occasions.

Digitized by Google

FRÉQUENCE ET VALEUR DE L'ADÉNOPATHIE TUBERCULEUSE TRACHÉO-BRONCHIQUE DANS LA TUBERCULOSE MILIAIRE DES ENFANTS ET DANS LA MÉNINGITE TUBERCULEUSE.

PAR MM. P. HAUSHALTER, *Agrégé à la Faculté de Nancy;*

ET

A. FRUHINSHOLZ, *Interne des Hôpitaux.*

SUR 78 autopsies de tuberculose miliaire chez des enfants de moins de 12 ans, 74 fois (soit 95 f. p. 100), la tuberculose des ganglions trachéo-bronchiques a été rencontrée; 67 fois il s'agissait de méningite tuberculeuse, sur lesquelles 64 fois l'adénopathie existait.

Cette adénopathie a toujours les caractères d'une tuberculose ancienne, d'une tuberculose caséeuse; dans 29 cas elle coïncidait avec des lésions tuberculeuses anciennes du poumon, généralement localisées; dans 44 cas elle réalisait la seule lésion tuberculeuse ancienne.

Dans la presque unanimité des cas donc, l'origine de l'infection bacillaire, qui aboutit chez les enfants à la granulie et à la méningite, réside dans la caséification des ganglions trachéo-bronchiques. La fréquence de cette lésion est une preuve de l'importance de la contagion aérienne de la tuberculose chez l'enfant.

Comment cette adénopathie trachéo-bronchique tuberculeuse, plus développée, plus profonde dans la tuberculose miliare que dans n'importe quelle autre forme de tuberculose, aboutit-elle à l'infection bacillaire sanguine qui est considérée comme presque l'unique cause de la granulie et de la méningite? Malgré les rapports étroits des vaisseaux de la base du cœur avec le paquet des ganglions caséeux du médiastin, rapports que montrent les planches que nous présentons au Congrès, nous n'avons pu déceler de lésions macroscopiques ou microscopiques des parois de ces vaisseaux, pouvant expliquer le passage direct des bacilles des ganglions dans le sang.

L'infection bacillaire sanguine se fait sans doute par l'intermédiaire du système lymphatique: pendant le très bref trajet effectué par les leucocytes bacillifères depuis les ganglions caséeux du médiastin jusqu'au torrent circulatoire, en passant par les lymphatiques efférents et une courte partie du canal thoracique ou de la grande veine lymphatiques, la destruction des bacilles n'a peut-être pas le temps de s'opérer.

Même dans bien des cas où la méningite existe pure, sans granulie

Digitized by Google

généralisée, et où on serait peut-être tenté, en raison de ce fait, de chercher l'origine de l'infection, non dans la voie sanguine mais dans la voie lymphatique, il est probable qu'il existe une infection bacillaire générale dont les manifestations sur les autres organes ne sont pas appréciables à l'œil nu, ou sont, comme nous avons pu le constater plusieurs fois, extrêmement discrètes.

THE RÔLE OF THE NASAL FOSSÆ IN THE PROPHYLAXIS AND TREATMENT OF PULMONARY TUBERCULOSIS.

By Dr. MAURICE MIGNON, *Nice*,

Late Assistant Laryngologist to the Deaf and Dumb Hospital, Paris.

When we consider the question of the prophylaxis of tuberculosis, we must recognise the fact that contagion takes place chiefly through the air. Air is the vehicle by which the microbes invade the organism far more frequently than foods, which can be sterilised by cooking. When the air is still infective, in spite of the use of spittoons, in spite of the practice of disinfection, in spite of every precaution intended to prevent the spread of the disease, the nasal fossæ are still capable of arresting the danger that threatens us. The microbes that enter with the air are, in a large measure, arrested by the cilia of the nasal vestibule and by the very extensive and very irregular surface of the mucous membrane. One may thus recognise the bactericidal function of the nasal mucus, although it has been questioned by some authors. Clinical experience teaches, indeed, that the nasal fossæ are much more resistant to tuberculosis than the rest of the respiratory tract, and even than the bucco-pharyngeal cavity. Insufficient nasal permeability (nasal obstruction from malformations or septal ridges, from hypertrophic or congestive rhinitis, from cysts, vegetations, adenoids, etc.), should therefore be reckoned among the dangers of tuberculous infection.

From the point of view of treatment the state of the nasal fossæ is of equal importance. As the nose allows more air to enter than the mouth, nasal insufficiency results in deficient oxidation of the blood, and everyone knows how necessary oxygen is to the tuberculous. Entering by the mouth the air brings with it harmful microbes, which, accompanied by dust, favour the malady. Moreover, this air, insufficient and injurious, is unmodified, either in temperature or in pressure; it provokes bucco-pharyngeal, laryngeal and tracheo-bronchial inflammations which impede the action of treatment.

It is therefore absolutely necessary that we should be satisfied that patients presenting themselves for examination (especially those disposed to tuber-

Digitized by Google

culosis, and those who are themselves tuberculous) are not suffering from any cause of nasal insufficiency. If any defect is present it should be remedied, and we should enjoin the patients to breathe solely by the nose as soon as they are able, for in this habit often plays a part. Instruction on the latter point should be included in the general advice which one makes a point of disseminating amongst all classes of the population.

LE RÔLE DES FOSSES NASALES DANS LA PROPHYLAXIE ET LE TRAITEMENT DE LA TUBERCULOSE PULMONAIRE ET LARYNGÉE.

PAR M. LE DR. M. MIGNON, *Nice*,

Ex-Assistant de Laryngologie aux Sourds-Muets de Paris.

APRÈS avoir constaté l'extension des ravages de la tuberculose, nous entrons dans une période de lutte à outrance contre elle, et nous ne devons épargner aucun des moyens qui s'offrent à nous soit pour éviter la maladie, soit pour la combattre.

Aussi croyons-nous l'occasion tout à fait favorable pour attirer l'attention sur un ensemble de faits dont se sont beaucoup occupés les rhinologistes, et qui méritent d'être pris en considération par tous ceux qui s'intéressent à l'étude de la tuberculose; nous espérons pouvoir en tirer des conclusions utiles pour la prophylaxie et le traitement de cette affection.

On sait que la contagion de la tuberculose se fait surtout par l'air; il est le véhicule du bacille de Koch, beaucoup plus fréquemment que les aliments pouvant d'ailleurs être stérilisés par la cuisson. Mais lorsque l'air est encore nocif, malgré l'usage des crachoirs, malgré les pratiques de désinfection, malgré toutes les précautions que l'on fait connaître pour empêcher la propagation de la maladie, les fosses nasales peuvent dans certaines conditions arrêter ou tout au moins diminuer le danger qui nous menace.

En passant dans les voies respiratoires supérieures, l'air se débarrasse d'une partie de son contenu, en même temps qu'il subit des modifications de pression, de température et d'hygrométrie. Poussière et microbes trouvent dans les fosses nasales de nombreux obstacles, qui en arrêtent la plus grande partie tout en laissant passer l'air. On s'en rend compte d'une façon très simple en comparant les mucosités nasales rejetées par une même personne selon qu'elle séjourne au milieu des agitations de Paris, ou qu'elle reste au contraire à la campagne par un temps calme. Dans le premier cas seulement, on constate que le mouchoir est souillé par d'abondantes poussières noires qu'il est inutile d'examiner au microscope pour les considérer comme

Digitized by Google

dangereuses. Des expériences ont été faites pour le prouver par Lannelongue, Achard, et Gaillard ;* la mort de cobayes tuberculeux a été hâtée par l'insufflation de poussières ; plusieurs des animaux ont succombé manifestement à des broncho-pneumonies. Ces expériences confirment les observations de la clinique ; chez l'homme l'inhalation des poussières paraît jouer un rôle important dans la genèse et l'évolution de la tuberculose pulmonaire.

En même temps que les poussières, les microbes contenus dans l'air respiré se déposent sur les vibrisses du vestibule nasal et, en moins grand nombre, à la surface de la muqueuse. C'est à cause de son rôle de filtre que l'intérieur du nez présente au voisinage de son entrée une flore microbienne aussi riche. Ainsi le prouvent les recherches de B. Fränkel, Hack, Strauch, Loewenberg, Netter, Hajek, Rémann, von Besser, Thost, Deletti, Paulsen ; on y trouve le bacille de la tuberculose assez fréquemment chez des personnes qui ne présentent aucun signe de la maladie (Straus †). Mais si, au lieu d'examiner les sécrétions du vestibule nasal, on recueille les sécrétions normales des parties profondes de la muqueuse, on obtient un résultat très différent.

D'après les observations faites par Würtz et Lermoyez,‡ le mucus nasal que l'on obtient par l'excitation d'une pituitaire saine ne contient généralement pas de microbes. Cependant parfois des bactéries pathogènes se rencontrent dans le nez d'individus sains. De même Thomson et Hewlett,§ Piaget‖ ont constaté la rareté des bacilles sur la membrane pituitare. D'autres auteurs, au contraire, Klemperer,¶ Malato,** ne partagent pas cet avis. Park et Wright †† disent aussi qu'on trouve constamment plus ou moins de bactéries dans l'intérieur des fosses nasales. Entre ces opinions, il semble très rationnel d'admettre que les microbes diminuent de fréquence à mesure qu'on pénètre dans la profondeur des fosses nasales, étant retenus en grand nombre par les surfaces humides qu'ils rencontrent au début de leur passage. A cette raison d'importance capitale peut s'ajouter l'action plus ou moins bactéricide du mucus nasal. En effet, les expériences de Würtz et Lermoyez, de Piaget, prouvent que le mucus nasal est un très mauvais milieu de culture. Le pouvoir bactéricide que lui reconnaissent ces auteurs n'est pas admis par Park et Wright ; ils appuient leur opinion sur deux ou trois essais infructueux, et surtout sur les faits connus de persistance de bacilles de Leoffler dans les fosses nasales de convalescents diphtériques (cas de Legendre et Pochon, Sevestre, Netter, etc.), ou dans le nez d'étudiants

* Lannelongue, Achard et Galliard. "De l'influence des poussières sur l'évolution de la tuberculose." 'Académie des Sciences,' 6 mai 1901.

† Straus. 'Ref. Centralbl. für Bakter,' 1895. Ed. I., p. 96.

‡ Würtz et Lermoyez ('Annales des maladies de l'oreille, du larynx, du nez.' Août 1893).

§ Thomson et Hewlett. ('Méd. chirurg. trans.,' vol. lxviii., 1895.)

‖ Piaget. ('Annales des maladies de l'oreille,' fév. 1897.)

¶ Klemperer. ('Ref. Journ. of laryngology,' nov. 1896, p. 286.)

** Malato. ('Archivio Italiano di otologia,' VI., fasc. 4, 1897.)

†† Park et Wright. ('Annales des maladies de l'oreille,' 1898, I., page 113.)

Digitized by Google

soignant des malades. Mais ces observations tendent, au contraire, à prouver que la virulence de ces bacilles pouvait être atténuée par le mucus, puisqu'ils ne se développaient pas. Pourquoi n'en serait-il pas de même du bacille de la tuberculose trouvé dans les fosses nasales de sujets indemnes, exposés à des poussières chargées de bacilles (Straus). Il nous semble que le mucus nasal a un pouvoir bactéricide relatif, suffisant pour lutter contre certaines infections (bactéridie charbonneux), insuffisant pour les empêcher toutes (streptocoque). Le bacille de Koch, tout en ne perdant pas complètement sa virulence, ne peut échapper complètement à cette influence, à laquelle s'ajoute l'action phagocytaire des leucocytes que l'on trouve dans les fosses nasales (Metchnikoff-Viollet*).

Notre hypothèse est du reste basée sur la clinique; les fosses nasales sont, nous le savons tous, beaucoup plus réfractaires à la tuberculose que le reste de l'arbre respiratoire, tout en étant les premières voies exposées à l'infection.

Toutes les recherches faites sur cette question le prouvent amplement (Cartaz,† Thorwald-Riedel, Raulin). Willigk, sur 476 autopsies de tuberculeux, a trouvé la tuberculose nasale une fois seulement; Bryson Delavan, contre 103 lésions bucco-pharyngées, ne découvre que quatre cas de lésions nasales. Ce caractère réfractaire de la muqueuse pituitaire à la tuberculose a même fait le sujet d'essais thérapeutiques: Fauvel‡ a traité des tuberculeux avec quelque succès par des injections d'extrait organique de muqueuse nasale de chevreau et de mouton (paratoxine T).

Pour que cette défense de l'organisme se fasse dans de bonnes conditions, il faut que la muqueuse nasale ne soit pas altérée par des modifications pathologiques; celles-ci, en même temps qu'elles empêchent sa fonction protectrice, provoquent des troubles favorables à l'infection tuberculeuse. On a souvent l'occasion de remarquer la relation qui unit le coryza et les troubles respiratoires.

D'après Freudenthal§ le catarrhe rétro-nasal doit être considéré comme un des points de départ de la tuberculose pulmonaire, et nos observations concordent tout à fait avec les remarques de cet auteur. Pendant un séjour de plus de deux ans à l'Asile de Convalescence de Vincennes, nous avons pu examiner et interroger de nombreux tuberculeux à des périodes diverses, et aussi assister au développement de cette affection chez un assez grand nombre de convalescents indemnes cliniquement au début de leur séjour.‖ Nous avons même eu la satisfaction de voir adopter, par le Congrès de 1898, le vœu que nous émettions, c'est-à-dire, qu'il ne soit point envoyé de tuberculeux dans les asiles de convalescence ouverts à d'autres catégories de malades.

---

* Viollet. ('Arch. internat. de Laryngologie,' janv.-fév. 1900.)

† Cartaz. ('France Médicale,' juillet 1887.)

‡ Fauvel. ('Congrès pour l'étude de la Tuberculose,' Paris, 1898, p. 901.)

§ Freudenthal. ('Annales des maladies de l'oreille,' etc., 1897, I., p. 287.)

‖ M. Mignon. "La tuberculose pulmonaire à l'Asile de convalescence de Vincennes." ('Congrès pour l'étude de la Tuberculose,' Paris, 1898, pp. 416 et 925.)

Digitized by Google

Tout en tenant compte de la prédisposition que leur affection récente donnait à ces malades, nous avons été frappés du mauvais état de leurs fosses nasales ; le catarrhe rétro-nasal y était, pour ainsi dire, constant, accompagné avec une fréquence toute particulière de lésions diverses entraînant plus ou moins d'obstruction.

Sans avoir fait de statistiques, dont nous connaissons tous le peu de valeur en pareil cas, nous ne pouvons donc souscrire à l'opinion de Fletcher-Ingals * qui, à l'encontre de Freudenthal, soutient que la tuberculose pulmonaire est plus rare chez les individus atteints de catarrhe nasal ; pour défendre ces statistiques compromettantes, l'auteur admet que le catarrhe nasal fait partie souvent d'un état hypérémique, tandis que la tuberculose se greffe sur l'anémie. Nous ne suivrons pas l'auteur dans ce raisonnement pourtant si discutable. Contentons-nous de souhaiter que dans tous les cas où des théories contradictoires sont émises, il soit aussi facile de se faire une opinion.

Du reste, la toux seule, par les troubles qu'elle provoque sur toute l'étendue des voies respiratoires, met déjà le sujet en mauvaise situation pour se défendre. Et chacun de nous connaît l'action des lésions nasales sur l'appareil respiratoire. Tantôt elles provoquent seulement de la toux, et en même temps une perturbation dans la respiration par suite du spasme glottique et bronchique (Ruault,† François Franck,‡ Gaston§) tantôt elles amènent des lésions de laryngo-trachéo-bronchite qui favorisent l'infection (Ruault, Cox,‖ Scheppegrell ¶).

L'ensemble de ces faits nous permet donc d'établir solidement notre opinion et de considérer, comme un danger d'infection tuberculeuse, le mauvais fonctionnement physiologique de la muqueuse nasale (catarrhe nasal, rhinite atrophique), ou l'insuffisante perméabilité des fosses nasales (rhinite congestive ou hypertrophique, déviations ou crêtes de la cloison, tumeurs kystiques ou autres, végétations adénoïdes, etc.).

Si au lieu de considérer le sujet sain, toujours plus ou moins exposé à la contagion, nous examinons la situation des malades déjà infectés, nous sommes obligés d'admettre qu'ils partagent les mêmes dangers que les gens bien portants, tout en offrant moins de résistance. De plus, l'état des fosses nasales a une influence sur l'efficacité du traitement qu'ils ont à suivre, et nous allons chercher à le démontrer.

La cure d'air a toujours été considérée comme ayant une importance capitale ; et l'on aura beau soumettre le malade aux meilleures conditions ambiantes, comment pourra-t-il en profiter, si les fosses nasales entravent la

---

* Fletcher-Ingals. ('British. med. journ.,' 1897.)

† A. Ruault. ('Archives de laryngologie,' 1888.) "Traité de Médicine," 2e édition, VI., p. 40.

‡ François Franck. ('Archives de physiologie,' juillet 1889.)

§ Gaston. ('Journal de Clinique et de thérapeutique infantile,' 18 déc. 1896.)

‖ Ch. N. Cox. ('Journal médical de Brooklyn,' octobre 1896.)

¶ Scheppegrell. ('New York Med. Journ.,' décembre 1898.)

Digitized by Google

respiration? L'air doit entrer dans les poumons en aussi grande quantité que possible pour favoriser l'hématose. On sait que dans un même temps, et sous un effort égal, le nez laisse passer une plus grande quantité d'air que la bouche ; les recherches de Mendel,* au moyen du rhinomètre, le prouvent par des traces pneumographiques. On ne peut donc espérer compenser par la respiration buccale une respiration nasale défectueuse ; nous n'ignorons pas du reste, les nombreux inconvénients de cette suppléance (pharyngite, laryngite). L'augmentation de la perméabilité nasale permettant l'absorption de plus d'oxygène, favorise la production de l'hémoglobine. Lichtwitz et Sabrazès† ont constaté qu'après l'opération le sang des adénoïdiens se modifie dans le sens de la formule normale ; il se produit une augmentation de globules rouges, et une diminution de globules blancs, la première étant due vraisemblablement à une meilleure hématose, la seconde à la suppression du tissu adénoïdien.

L'insuffisance de l'oxygénation n'est pas le seul inconvénient qu'entraîne l'obstruction nasale ; elle empêche l'air inspiré de subir d'utiles modifications. La muqueuse nasale, en effet, agit sur la température et l'humidité de cet air, souvent trop froid ou trop sec ; elle en change la pression pendant l'acte respiratoire, à condition qu'il puisse suivre librement son trajet normal ; tout en le débarrassant des principes nuisibles, dont nous avons déjà parlé (microbes-poussières), elle produit dans sa composition des échanges chimiques non négligeables. Goodale‡ a étudié les variations de chaleur, d'humidité, de pression, de trajet, produites sur l'air de la respiration dans les diverses affections des fosses nasales. Il en résulte des changements importants, qui expliquent, en dehors de l'action microbienne, les affections formant le cortège de l'insuffisance nasale, c'est-à-dire, le catarrhe naso-pharyngien, l'inflammation bucco-pharyngée, laryngée, trachéo-bronchique (Ruault, Cox, Scheppegrell), et même l'emphysème provoqué d'une façon expérimentale par Cousteau.§

Telles sont les conséquences variées de l'insuffisance nasale ; il est bien évident que si elles agissent sur l'individu normal en diminuant sa résistance à la contagion, elles ont une influence encore plus funeste chez le tuberculeux, dont la première partie du traitement consiste à absorber le plus d'oxygène possible, et à éviter toute cause pouvant amener un état inflammatoire de l'arbre respiratoire.

Maintenant que nous avons établi le rôle pernicieux de l'insuffisance nasale, une question se pose. Quand peut-on dire qu'il y a insuffisance nasale? Évidemment, c'est là une question d'appréciation. A côté de l'obstruction complète (congénitale ou due à la présence de tumeurs, d'adhérences, etc.), il reste des cas beaucoup plus nombreux où elle est

---

* Mendel. "Physiologie et pathologie de la respiration nasale." Paris.

† Lichtwitz et Sabrazès. ('Arch. int. de laryngol.,' janv.-fév. 1900.)

‡ J. L. Goodale. ('Boston med. and chirurg. journal,' nov. 1896.)

§ Cousteau. ('XIII[e] Congr. intern. de Méd. Section de laryngologie.' Paris, 1900.)

Digitized by Google

seulement relative (rhinite hypertrophique, déviations ou crêtes de la cloison, végétations adénoïdes, etc.) ; d'autres fois, les fosses nasales ne remplissent pas leurs fonctions, soit parce qu'elles sont trop larges, soit surtout parce qu'elles sont le siège de modifications pathologiques (rhinite atrophique, infections nasales). Nous ne croyons pas nécessaire d'apprécier avec exactitude la perméabilité du nez et d'employer pour examiner un sujet à ce point de vue soit un rhinomètre, soit l'une des méthodes préconisées par Zwaardemaker, Rayser, ou Jacobson.* On pourra en général résoudre la question facilement en appréciant la façon dont pénètre et sort l'air de la respiration, et surtout en examinant au speculum et du stylet l'état des fosses nasales. Cet examen est d'autant plus utile que beaucoup de personnes, respirant bien d'un côté, respirent très mal de l'autre, et ne s'aperçoivent d'aucun trouble ; le médecin ne doit donc pas attendre qu'un malade se plaigne de gêne respiratoire pour l'examiner à ce point de vue, s'il s'agit d'un prédisposé à la tuberculose, et à plus forte raison d'un tuberculeux.

Nous croyons même qu'il serait important d'indiquer au malade l'utilité de respirer exclusivement par le nez, en raison du rôle que peut avoir l'habitude ; il est désirable que cette recommandation soit répandue dans tous les milieux sociaux en même temps que celles concernant l'usage des crachoirs, de la désinfection, etc. L'ignorance seule du danger peut favoriser la contagion. Quand la respiration nasale est difficile, le médecin doit juger si elle est insuffisante, et traiter les lésions presque toujours curables qui l'entraînent. Nous terminerons en rappelant le vieux proverbe, cité par Lermoyez, à propos du rôle physiologique des fosses nasales : " Ferme la bouche, sauve ta vie." La tuberculose est un danger si grand pour l'humanité que nous ne devons rien négliger pour tâcher d'abord de l'éviter, ou, faute de mieux, pour augmenter les chances de sa guérison.

---

*Jacobson. ('XIII^e^ Congrès intern. de Médecine. Section de Laryngologie,' Moscou, 1897.)

Digitized by Google

THE USE OF DRUGS IN PULMONARY TUBERCULOSIS.

By W. R. HUGGARD, M.A., M.D., F.R.C.P.,
Davos Platz, Switzerland.

In regard to the use of drugs in pulmonary tuberculosis there is much difference of opinion amongst medical men. By some physicians drugs are considered either useless or harmful; by others one drug or method of treatment is considered *the* treatment. Creosote, cinnamate of soda, arsenic, formaldehyd, and various other drugs have each their devoted adherents, who regard their pet remedy almost as a specific. A large number of medical men, while not discarding drugs altogether, employ them only for the relief of urgent symptoms.

All these views are in my opinion too narrow; some over-estimating the power of drugs, and others over-estimating their limitations. From another standpoint also the foregoing views are too narrow. The great variety in the clinical forms of phthisis is dropped out of sight; and the same line of treatment in regard to remedies is sometimes adopted without sufficiently precise reference to the case in hand.

The object of this paper is to point out some of the advantages that may be gained by the judicious use, and some of the evils that arise from the injudicious or indiscriminate employment of drugs in pulmonary tuberculosis. The broad principles will also be indicated which determine my own selection of remedies according to the variety of the case. The subject is one in which the comparison of individual experience is of the greatest value in extending our knowledge, and in putting it on a firmer basis.

The first point to be made clear is that up to the present no specific has been found for tuberculosis. Not merely so; but there is no drug that is useful in all cases. Nevertheless there are various drugs that favourably influence the course of some varieties of the disease always provided that they are well tolerated by the patient, and that they do not cause any disturbance of appetite, digestion, or assimilation.

Time does not permit even a hasty review of all the drugs useful in phthisis. The various conditions only that indicate or contra-indicate one remedy rather than another will be rapidly considered.

What medicines have we at our disposal? Drugs may favourably influence the course of the disease—

1. By improving the general health; as, for example, arsenic, quinine, strychnine, lime, phosphorus preparations.
2. By increasing the local resistance of the affected tissues, as creosote and its derivatives, salicyl preparations, and counter irritants.
3. By modifying the quantity or character of the secretions, as the balsams, the terebinthinates, the essential oils, morphine and apomorphine, and inhalations, especially of formaldehyd.

Digitized by Google

4. By controlling symptoms that react prejudicially on the patient. Amongst these are :—

   a. Digestive ailments.
   b. Excessive or needless cough, which shakes and exhausts the patient, causes fever, or prevents sleep.
   c. Scanty expectoration and retention of secretions.
   d. Fever, which spoils appetite, and prostrates the patient.

5. By removing complications, such as syphilis.

In the choice of remedies the points to be considered are not the same as guide us in diagnosis or in prognosis. The first and most important point to recognise is, that treatment by medicines must be altogether subordinate to general hygienic management—good food and fresh air with rest and exercise according to the individual needs of the patient.

The first and most important point in treatment is to consider the state of the digestive system. If the stomach or digestion is out of order drug treatment is, as a rule, inadvisable, except in so far as it may aid in restoring the digestive functions to a normal state, or in removing some condition that tends to prolong the digestive ailment. For example, appetite and digestion not uncommonly improve with the artificial reduction of temperature, or with the control of excessive cough. In such cases we must feel our way cautiously and be guided tentatively by the results.

The most important indication then is to get the stomach and bowels into proper working order. Nothing goes right so long as the digestion is wrong.

Temperature is the next most important guide to treatment. Pyrexia will in a large proportion of cases yield to absolute rest, bodily and mental, combined with life in the fresh air. In many cases, however, a return to a normal level of temperature can be hastened by the administration of small doses of phenacetin—one to three grains—in combination with quinine and salol and sometimes arsenic. Taken in this way phenacetin seems to have rather a tonic than a prejudicial effect on the heart and on the general health, and may without hesitation be continued for months if need be. My experience of other antipyretics, save for quite occasional use, has not been so favourable, with the exception perhaps of salipyrin.

Arsenic, strychnine, quinine, and salicyl preparations greatly diminish, in my opinion, the tendency to recurrent attacks of subacute inflammatory character and those recurrent febrile attacks without any change in the physical signs which are so marked a feature in a large number of patients suffering from pulmonary tuberculosis. These recurrent febrile attacks are no doubt of very various origin, tubercle as a rule rendering the organism highly sensitive to influences that cause pyrexia. The drugs I have mentioned seem to me to diminish this sensitiveness to febrile reaction. In doing so they accomplish something more than the avoidance of a temporary drawback or inconvenience. In tuberculosis, more than in any other disease, slight drawbacks are apt to provoke further prejudicial effects, and each morbid condition

Digitized by Google

tends to become chronic. In averting slight drawbacks therefore we avoid great dangers which might completely alter the course of the disease. The disease is one in which pre-eminently anything short of the best is bad.

Next to pyrexia, as a guide to treatment, I would place a marked tendency to haemoptysis. Creosote and guaiacol increase, in my opinion, the liability to pulmonary hæmorrhage—as well, in fact, as to pulmonary inflammation—and should be avoided where such a disposition is present. The lime salts, the terebinthinates, and the balsams have, I believe, on the contrary a somewhat restraining influence, as has also morphine in minute doses.

The treatment of excessive cough requires much judgment. A closely allied object of treatment is the modification and, as a rule, the diminution of bronchial and pulmonary secretion—the "drying up of the lungs." A certain amount of cough is in a large number of cases indispensable, and has a salutary influence. The problem is to secure the removal of the pulmonary and bronchial secretions with the least amount of violence, exertion, or fatigue for the patient. Sometimes the secretions are extremely abundant, sometimes too scanty. The balsams and the terebinthinates and tar for the most part diminish bronchial secretion, and the terebinthinates commonly render it at the same time less tenacious and easier to get up. Minute doses of morphine —from one one hundred and twentieth to one sixtieth of a grain of any of its salts—diminish secretion, but as a rule render it more tenacious. Apomorphine in small doses—one-twentieth to one-sixteenth of a grain of the hydrochlorate—loosens the secretion without making it much more abundant. The recently-introduced morphine derivatives, heroin and dionin, greatly diminish cough, and to some slight extent diminish expectoration, while they are free from the drawbacks that morphine has, of rendering the secretion viscous and difficult to get up. They are also practically devoid of constipating effect, and they do not upset the digestion. For the last two or three years they have almost altogether replaced in my practice the phosphate of codeine, which has fewer drawbacks than has morphine for the relief of excessive cough.

The most valuable agent that I am as yet acquainted with for modifying the bronchial and pulmonary secretions is the vapour of formaldehyd. For the last three years I have used this drug extensively, and with more and more satisfactory results. When steadily used it generally causes the secretions to be less purulent and more mucous, at the same time diminishing the amount and rendering expectoration easier. Where the use of the drug has been steadily persisted in for months tubercle bacilli have, as a rule, also become much less numerous, and in some old cases even have disappeared. The mode of employment requires a little care. The best way, in my opinion, to use formaldehyd is by means of a muzzle inhaler. The strength of the solution should at first not be more than two or three per cent. in rectified spirits of the ordinary forty per cent. solution. The addition of some essential oils renders the inhalation quite agreeable. Only from five to ten minims at first should be put on the inhaler, and this quantity should be renewed every

Digitized by Google

fifteen or twenty minutes, the entire time of inhalation being from two to four hours a day. The strength may be gradually increased up to six or eight per cent., and sometimes even to double that amount. If cough is very irritable, chloroform may be added to the inhalation. From independent observations I am strongly of opinion that chloroform, apart from its soothing influence on the cough, has in some cases a beneficial influence on the disease. It must, however, be used with judgment. I have known it seemingly give rise to pyrexia, by causing retention of secretion. The inhalation of formaldehyd on the contrary tends considerably to diminish pyrexia due to the absorption of toxins. Formaldehyd has another advantage. If not too strong, it diminishes irritability in the pharynx and larynx. It is, however, very irritating to the eyes and nose, and for this reason should be used only with an oral inhaler, or if the Burney Yeo oro-nasal inhaler is employed, it should be placed below the nose.

According to my experience syphilis is a complication of pulmonary tuberculosis in a much larger proportion of cases than is usually thought. In such cases I consider the treatment of the syphilis to be of the first importance. Small doses of the perchloride of mercury have then, instead of a depressing, a remarkably tonic influence. Intra-muscular injections of mercurial salts, though more strikingly beneficial, have as a rule the drawback of being excessively painful. To this rule, however, *huile grise*, or mercurial ointment rubbed up with sterilised oil, is an exception. The iodide of ethyl by inhalation is perhaps the least objectionable, while not the least efficacious mode of giving iodine. It may be combined with the formaldehyd inhalation.

To sum up, then, my line of treatment is determined, in the first instance, by the digestive system, by the general health, and by the state of nutrition.

If digestion is bad, the only drugs indicated are such as will restore it to a normal condition. If digestion is good, the general condition satisfactory, and the patient improving, I refrain from using drugs unless some definite indication is present. Amongst the most important indications for drugs are persistent afternoon pyrexia in spite of absolute rest out of doors; a tendency to recurrent febrile attacks or to slight inflammatory attacks. This tendency is usually combined with impaired nutrition and with a low state of general health. In these conditions arsenic, strychnine, quinine, and salol are amongst the most useful tonics. Active softening, excessive cough, over-abundant expectoration, and, more rarely, scanty expectoration, expectoration of extremely purulent or nummular character, require attention; and the drugs useful for these conditions generally seem to me to have a favourable influence on the course of the disease in addition to the temporary relief from discomfort they may afford. Amongst such drugs are formaldehyd vapour, creosote and its derivatives, except the carbonate of guaiacol, which has generally seemed to me to be inert, terpene hydrate, oil of cinnamon, myrtol, the balsams, and the lime salts. In sluggish chronic softening, counter-irritation by iodine or by small flying blisters is of the greatest use. When an old syphilitic taint is present its removal should be our first care.

Digitized by Google

The short time at my disposal does not permit a detailed examination of the points I have brought forward, and my remarks are rather to be considered in the light of heads for discussion than as even a partial exposition of the subject. What I have said may, however, I trust, be sufficient to induce others to bring forward their experience on the same lines.

REMARKS ON TUBERCULOSIS AND ITS TREATMENT.

By Dr. BARADAT,
Consulting Physician at Cannes, French Riviera.

In the case of tuberculosis, as in that of every infectious disease, two factors must be taken into consideration.

The first of these is the infectious agent, the morphological and biological characters of which are so well known nowadays; the second is the soil which the agent has developed itself in, and whose characteristics are either acquired or hereditary.

All rational medication must, to be complete and really efficacious, apply to these two factors, and take into account all the elements which arise in a given case. For, as Leudet says, tuberculosis presents, in its varied manifestations, special idiosyncrasies differing absolutely from one individual to another.

Under these conditions only can we hope to be victorious over this dread disease.

As a matter of fact, a review of the new methods of treatment employed in dealing with tuberculosis reveals to us the fact that, although these methods are, without doubt, of real value, they are only efficacious against certain given systems, and possess no influence over the whole of the phenomena which are to be overcome; certainly, they have special indications, but they are insufficient, because their field of action is but a limited one.

Amongst these indications, the comparative effects of which we shall examine later on, some are destined to improve and to strengthen the soil, others, on the contrary, are specific agents; they give rise to the diapedesis of the white globules, thus multiplying the means of defence with which the organism is provided in its struggle against the bacilli.

A thoroughly rational treatment should take both factors into account, that is to say, the medication employed should act in two ways, both as a dynamogenetic agent and as a specific. One is generally inclined to look upon each new method of treating tuberculosis as one that will immediately effect a radical cure of this terrible disease, without taking into account either the infectious agent and its toxine, or the soil on which these latter react.

We must oppose this tendency, and attempt a true, careful, and impartial appreciation of the new medications.

Let us, for instance, take the case of an anæmic patient: the Koch bacillus has invaded his organism, but still remains latent; if we leave this

Digitized by Google

patient to himself his anæmia will increase, his digestive activity will diminish, his strength dwindle away, and assimilation will be reduced to a minimum; there will be, as has truthfully been said, a failure of the whole organism.

What must be done to meet such a case?

Firstly, the organism must be strengthened, nutrition favoured; it is here that a use is found for medications tending to produce these effects, such as arsenic in its more easily assimilable forms (cacodylates), tannin, iodine, cod liver oil, salt lotions, alcohol frictions, sea-baths, a hygienic treatment.

By these means the bacilli will be kept under, their action neutralised, and as long as an equilibrium is maintained between the means of defence and the attack, the patient will live.

But a fatal time will come when the bacilli will gain the upper hand, and this under the influence of varied causes, to which an organism already infected will have to pay a large tribute, such as physiological troubles, grief, repeated bronchitis, influenza, measles, scarlatina, and, especially in the case of young subjects, intense physical and intellectual strain—too much bicycling, too much fast living, an excess of emulation and rivalry in examinations and competitions.

So that this treatment of the soil, if we may be allowed this expression, which seemed at first so efficacious, had but an ephemeral effect; enough had not been accomplished, the disease should have been attacked in its very essence, the bacilli and their toxines destroyed.

It is the same with all medications in the case of tuberculosis, and I should willingly call them partial medications.

Let us consider those that address themselves to the soil, the constitution of the subject.

Firstly, we hold that a hygienic treatment should be the basis, the indispensable foundation-stone of every medication; without it, they will all fail.

As Professor Letulle so picturesquely puts it, the patient must be "*centrifuged*," he must be taken away from large towns, from the centres where diseased persons are collected; he must be given the pure, fresh, invigorating air of the seaside or of the mountains; he must have in profusion sunlight, an agent as salutary to man as it is destructive to microbes.

In our opinion, this hygienic treatment will best be realised by means of *free sanatoria*, Landouzy's home sanatoria, such as we find them scattered, in the shape of villas, along our sun-bathed Mediterranean shores. There all the required conditions, not only hygienic, but moral and inspiriting as well, can be fulfilled.

In private sanatoria for the rich, the culinary arrangements for such a large number of people are necessarily unsatisfactory, the cooking is less carefully attended to, the dishes less carefully prepared, and less adapted to individual wants, to stomachs often fatigued and upset. As a matter of fact, the question of food is of vital importance in the treatment of a disease in which superalimentation plays such an important part.

The private sanatorium should be reserved for the impulsive, for those who are incapable of energy and self-direction.

Digitized by Google

Besides, how many of these sanatoria are carelessly conducted! How many paying sanatoria are under the control of commercial managers, who allow alcohol in all its forms to be freely distributed! who close their eyes to promiscuities which are dangerous, often immoral and always harmful to patients who must carefully husband their strength.

On the contrary, we willingly acknowledge the usefulness of the sanatorium destined to the poor. In their case, hygienic discipline will always be maintained, for there will be no reason for unbending before the perspective of rapid gains and big dividends; on the other hand, the poor will always find at a sanatorium better feeding than at home.

As for the medical treatment, much has been said of cacodylate of sodium.

We shall not attempt a complete study of this substance. The most important thing for us is to be thoroughly acquainted with its real value. Its action and its efficiency must be measured by the light of the experience of numerous observers, and of our own. Its promoters were wrong, in our opinion, to call cacodylate of sodium a specific agent against tuberculosis. As against the numerous favourable observations, and which we do not doubt in the least, of Messrs. A. Gauthier, Renaut, Rendu, Letulle and others, we have to set off many others, equally unimpeachable, and where the results on tuberculosis have been *nil.*

In the course of our practice, we currently employ cacodylate of sodium; its effects have proved excellent in cases of anæmia, of ganglionic and lymphatic persons, of chlorosis; in such cases, we have observed a regular revival of the physiological functions, an increase of appetite, a resorption of ganglia. On the contrary, we have obtained less favourable results in cases of ulcerous and cavitary tuberculosis.

Burlureau, in a recent and thoroughly complete study on cacodylate, has come to the same conclusion. "As for tuberculosis," he says, "I regret to have to say that, contrary to the opinion of Professor Gauthier, it is in this disease that cacodylate has given me the least favourable results. Out of twenty-nine cases of pulmonary tuberculosis, I have only once obtained a really favourable effect, and that was but temporary."

Cacodylate will be specially useful for the predisposed, for those incipient cases which were so difficult to diagnose, and which we have now learned to recognise.*

As for the vanadates, they have not fulfilled the expectations formed of them; but this is partly due to the difficulty experienced in obtaining thoroughly determined products.

---

* For instance, Landouzy, Grancher, Sanchez have revealed to us the delicate stethoscopic signs of the period of germination; Bard and Faisans have showed us the importance of the cardiac rhythm, of tachycardia; Roussel and Boix that of the scapulo-thoracic amyotropia; Bouchard, Béclère, Kelsch, Maragliano have taught us the radioscopic signs of incipient tuberculosis; Arloing, Mongour, Courmont have established on a sure basis the early diagnosis of tuberculosis by sero-diagnosis by agglutination; Albert Robin and Binet give us the same certitude by the analysis of the respiratory chemism; Sirot and Fink by the observations of the effects produced by injections of artificial serum; Gaube (of Gers) by the study of the demineralisation of the tissues.

Digitized by Google

The same must be said of certain artificial serums, which must be classed among the soil strengtheners, and are wanting in bactericidal powers, or rather in the power of exciting diapedesis and phagocytosis.

We now come to the raw meat treatment.

The experimental researches of Richet and Héricourt have proved that raw meat juice acts, not as a strengthening agent, but as an antitoxine. This antitoxine would neutralise the effects of the tuberculosis toxine.

This juice is the muscular plasma, obtained either by the press or by congelation followed by rapid thawing of the muscular tissue.

The following is the method I have adopted at Cannes in the case of patients whom I submit to this treatment:—The daily quantity of mashed meat is 800 grs. (about 28 oz.); the patient takes as much as he can, the rest of the meat is pressed, in order that the juice may be extracted.

The plasma must be taken immediately after having been prepared, otherwise one risks swallowing a putrefied and toxic substance.

Although this method has given me excellent results, I consider it difficult to put into everyday practice.

It possesses many inconveniences. For one thing, it is not within everybody's reach; it is costly in preparation, and requires 800 to 1500 grs. of meat daily; it is supported with difficulty by many patients; it requires constant supervision, for this meat juice soon putrefies and becomes toxic. Injected under the skin of an animal, it causes death in a few minutes.

Experiments with this anti-tuberculous plasma have been made in the laboratory of Messrs. Richet and Héricourt. These attempts at hypodermic injections of an immunising and even curative liquid led us to read once more the already old but very complete works on the bactericidal or antitoxic properties of the blood of animals that are refractory, or seemingly refractory, to tuberculosis.

The medical literature of 1890 to 1895 shows us how this question has been strenuously discussed and deeply criticised.

However, from these works we glean the following fact—that the blood of certain animals confers on other animals immunity from tuberculosis, and may even cure this disease. "As far back as 1888," says Professor Bouchard, in writing to M. Bertin, one of the promoters of anti-tuberculosis sero-therapy, "I expressed the idea that vaccines were destined to play a part, not only in the prophylaxis, but also in the treatment of this disease."

For my own part I have no hesitation in attaching myself to the method of anti-tuberculous sero-therapy introduced at about the same time, in 1889, by Richet and Héricourt, and by Bertin and Picq, for I think that therein lies the solution, so long sought, of the problem of the cure of tuberculosis.

Naturally, with this medication just as with any other, we must not wait for the patient to be emaciated, to present digestive troubles and cachexia before treating him.

For—we insist on this point—tuberculosis is not consumption. A consumptive or phthisical person is one in whose case the Koch bacillus, after having terminated its progressive career, has slowly brought on the

Digitized by Google

suppurative destruction of the cells attacked; and in this mass of destroyed matter you will find all the processes provoked by the staphylo, the strepto, and the pneumo coccus working together with the Koch bacillus.

In this case, you have phthisis, consumption, the hectic fever which brings on a fatal issue; imagine that, by some means, you could at this period destroy the bacilli of tuberculosis, your patient would still succumb to the strepto-, the staphylo-, and the pneumococcus.

As a matter of fact, says Landouzy, it is this idea of helping those who are in the incipient stage, at a time when the germs of secondary infection have not yet attacked them, that has led medical men to make use of "the immunising agents that are anti-toxic or bactericidal owing to their strengthening action on phagocytosis."

This science of sero-therapy, which we owe entirely to the French school, has been perfected by Pasteur's most renowned disciples, by Drs. Duclaux, Roux, Grancher, Nocard, Metschnikoff, Yersin, Calmette, Leroux, Charrin, Marmorek, Boinet, and many others, among whom we must mention Bertin and Picq, who were, together with Richet and Héricourt, the promoters of modern sero-therapy.

The use of natural serum has given me unexpected results in serious cases of tuberculosis, and I have always been surprised to find that this method of treatment is not better known; natural serum seems to me to fulfil all the required conditions, for it is both dynamic and bactericidal.

As we know, in the case of tuberculosis, the bacillus acts as a destructive force, but its action is strengthened by that of other destructive forces due to the soil. In one case it will be anæmia, in another heredity, in another influenza, or intellectual or physical strain.

So, with these generalities, what are the characters required of a therapeutic agent against tuberculosis? We admit the stimulating and regenerative properties of the general tonics, cacodylates, phosphates, cod liver oil, etc.; we will even allow the antitoxic property of meat juice, but has anyone the right to say that each of these agents fulfils the two conditions necessary to the cure of tuberculosis? Certainly not, for they are either simply stimulating and strengthening, or simply antitoxic. Bertin and Picq's serum (goats' serum) seems to me, on the other hand, to be at the same time tonic, antitoxic, and bactericidal; it is the one we make use of.

As a matter of fact, daily experience tends to prove that every serum is dynamogenetic, and therefore a general strengthening agent.

This is proved daily by the use of artificial serum in the case of serious hæmorrhage, of anæmia subsequent to chronic diseases, of traumatic shock consecutive to operations. In taking into account, however, the comparative value of the two serums, natural and artificial, we find that a very small quantity of the former produces an intense therapeutic effect, whilst the same effect can only be obtained by employing a double or even triple dose of artificial serum. There is here a *quid divinum*, due evidently to the intimate composition of natural serum. No one nowadays denies the dynamogenetic action of serum—it is a recognised fact.

Digitized by Google

All we have to do is to repeat this action as often as required in order to maintain to a remarkable degree the resistance and the vitality of the patient.

Whilst awaiting experiments destined to throw light on the still obscure question of the mode of action of serums, we give preference to the theory propounded by Metschnikoff, who looks upon them, not as antitoxic, but as stimulating agents of phagocytosis, in other words, as *stimulines*, provokers of organic resistance.

Therefore, as we admit that the microbial destruction and the arrest of infection are due to phagocytosis, the aim of our therapy must be to increase the activity of the phagocytes, in order that they may the more easily accomplish their mission. Moreover, the happy results that we have obtained this winter by means of natural sero-therapy lead us to believe that this is the real and only effective method of realising the cure of tuberculosis, especially in its early stages, now that the means of diagnosis which we possess permit us to discover the very earliest symptoms of incipient tuberculosis.

This treatment is absolutely innocuous and easily applied; one hypodermic injection of two cubic centimetres every other day. In some cases, however, in the case of nervous patients especially, I have observed after each injection an exaggeration of cellular activity, showing itself in the shape of fever, erythema, and dyspnœa; in such cases I administer the serum internally. But, in order to obtain the same tonic and stimulating effects, I have to increase the dose, and administer ten cubic centimetres instead of two, as in the case of hypodermic injections.

These results agree with those obtained by Grasset, who concludes by saying that the administration of serum internally is the method of choice, because it is free from danger and gives rise to no accidents. Nevertheless, even with Bertin's serum, we are of opinion that the cacodylate medication should be employed as a precious adjuvant in most cases on the same level as tannin, iodine, and cod liver oil.

THE TREATMENT OF PHTHISIS BY INTRA-TRACHEAL INJECTION OF LARGE QUANTITIES OF IZAL.

By COLIN CAMPBELL, M.R.C.S., etc., *Saddleworth, Yorks.*

"Klebs" (quoted in *Brit. Med. Journ.*, July 6th, 1901), "impressed with the uncertainty of the treatment of tuberculosis in sanatoria, recommends the continuance of attempts to arrive at a satisfactory causal treatment."

Professor Koch, speaking of the treatment of consumptives in German sanatoria, says: "Compared with the great number of consumptives, the success of the establishments in question seems so small that a material influence on the retrogression of tuberculosis in general is not yet to be expected of them. . . . I do indeed believe that it will be possible to render sanatoria considerably more efficient. . . . But even then, and even if the

Digitized by Google

number of sanatoria be greatly increased, the total effect will always remain but moderate." (Address to Tuberculosis Congress, July 22, 1901.)

The opinions, above quoted, of two of the most illustrious "scientists" of the day should cause some hesitation on the part of those who insist on the all-sufficiency of the "open air treatment of consumption." The views expressed are not those of hostile critics, but of the friends, almost apostles, of the system, yet already (whilst sanatoria are only beginning to boom in Great Britain) they are doubtful. "From the reports hitherto issued," says Professor Koch, "as to the results that have been achieved in the establishments, we learn, further, that about 20 per cent. of the patients that have tubercle bacilli in their sputum, lose them by the treatment there."

Twenty per cent. of cures means 80 per cent. of—well, non-cures. I do not remember exactly what percentage of cures were claimed ten years ago for Professor Koch's "Tuberculin." I fancy it was nearer 100 per cent.; and yet the tubercle bacillus lives, and countless thousands die of tuberculosis.

The foregoing (Dr. Koch's address was delivered since the main part of this paper was written) justifies an attempt to draw back the attention of the medical profession to a system of treatment that is based on a bed-rock of common sense, the objective of which is, *the destruction of that common enemy of mankind*, the tubercle bacillus, *in its hatching place*. If that be practicable, who will deny that it is a common-sense method? And who is there who can tell of any living creature that is destroyed by open air? Can it be asserted that the tubercle bacillus in a case of lupus of the face is killed by open air? And if it cannot, on what reasonable grounds can it be believed that the same microbe *in the lung* can be killed by open air? It is like the case of certain specialists who claim that they can "cure" internal, invisible tumours by electricity. The treatment, however, fails when the tumour happens to be situated on the nose, or in some position where the results are obvious.

But the human mind ever craves after the miraculous, and those who supply the want will ever be regarded as prophets. By such process "disease" ever has been and ever will be "cured," but they are always "diseases" of the non-obvious class. Submit an obvious cancer, or "lameness" resulting from an amputated leg, to the most up-to-date of miracle waters, and it is a million to one against the "cure."

It is more than seven years since Mr. Walter Whitehead presented my paper on "The Treatment of Respiratory Affections by means of Large Medicinal Injections through the Larynx," to the Royal Medical and Chirurgical Society ("Transactions," vol. 78). That paper described all previous published work on the subject, as well as two years of my own experience of the method, with details and results obtained in twenty-five cases.

Up to the time when I first began to systematically treat pulmonary affections by what I may be permitted to call "The Direct Method," all the cases reported had been treated by injections of solutions in olive oil.

The late Sir Thomas Grainger Stewart reported in the *Brit. Med. Journ.*, 1892, two cases of bronchiectasis treated in this way—successfully. Just then

Digitized by Google

I had a case of bronchiectasis, which I treated with oil solution. I referred to it as follows :—

"It was a case of old-standing disease—one of the earliest cases treated by me. Having Sir T. Grainger Stewart's Paper before me, I expected good results. One drachm, and later 1½ drachms, of a solution—4 per cent. guaiacol, 12 per cent. menthol, in olive oil—were injected twice daily for a month, but no beneficial result followed. Observation of this case first suggested to me that olive oil was not the best vehicle in which to convey the medicinal agent to the lung. A demonstration of this was easy. I collected some of the dense expectoration from the case in a U-shaped test-tube, closed with a cork at one end, and poured some of the oily solution on to it; after twenty-four hours the oil still remained at the top, whilst the lower stratum (which could be smelt by removing the cork) did not appear to contain either menthol or guaiacol. A glycerine solution at once suggested itself. My first experiences with glycerine solutions were, however, quite unsatisfactory. I could only note that *in a test-tube* it mixed better with expectorated material, and therefore presumably ought to bring the drugs (if it would only carry them) into closer contact with the diseased parts.

"It is all very well to demonstrate, as Sehrwald and Reichert have done, that the injected material can be found all through the lung tissues after an injection. So could it be found in the bladder. A few minutes after an intra-tracheal injection of turpentine I have noticed the characteristic smell of violets in the urine.

"The simple tube experiment mentioned suggests that the finding of menthol in the bronchioles, &c., after the injection of an oily solution (not miscible with the bronchial secretions) is more probably due to a secondary effort at *excretion* (subsequent to absorption) so easily noticed in the kidneys; and this, although probably very beneficial, is, I take it, quite different from bringing the original strong solution into *direct contact* with the diseased parts.

"That glycerine is the best solvent for remedies directed to pulmonary phthisis, bronchitis, or asthma, I have no doubt; but for months I did not succeed in finding a satisfactory solution. It is unnecessary to go into detail. I found that the ordinary 'glycerines' are most irritating to the bronchial membrane. Price's distilled glycerine is not so. Menthol, guaiacol, creosote, benzosol, &c., can be kept in solution in glycerine by heat." ("Trans. Roy. Med. Chi. Society," 1894.)

Thus it will be noticed that so long ago as November, 1894, I advocated glycerine instead of oil as the best vehicle. But oil has held the field, and has done worse than merely prevent success; it has caused the method to be discredited. In his article on "Bronchiectasis," in Allbutt's "System of Medicine," Dr. Ewart mentioned my work on this subject (vol. v., 1898), but only to condemn—on erroneous grounds. For the only solution or medicament mentioned by him is Sir T. Grainger Stewart's menthol-guiacol-oil-solution, which I had condemned four years before, and Dr. Ewart says in conclusion: "From personal observation I regard the use of the method (intra-laryngeal

Digitized by Google

injections of oil) in cases of phthisis with grave suspicion; and it has been my regret to witness, after its repeated use, the appearance of persistent râles of bronchial engorgement, and of catarrhal pneumonia of both bases, in a case which ended fatally." This was exactly my experience of it in 1892. Oil blocks the bronchioles; injections of oil suffocate.

Even so late as July 10th an eminent London specialist assured me that he had never heard of anything other than oil solutions being injected *viâ* larynx! There are, however, other objectors than those whose experience of oil solutions have proved so dangerous. The vast majority of those members of the medical profession to whom I have endeavoured to explain the obvious advantages of the "direct method" have refused to believe such a method practicable or possible, and when I have invited them to come and see it done with their own eyes, my invitations have invariably been declined with thanks.

Assuming now that it be admitted that it is possible to inject one or two, or even three ounces of fluid at a single sitting, I am to-day able to enlarge very considerably on my knowledge and experience of 1894. The solutions I then employed were most useful, if not demonstrably curative, by clearing the lungs out. Glycerine alone causes a greatly increased flow of pulmonary secretions (checked or blocked up by oil injections), and combined with one or other of the creosal compounds, notably benzosol, or with menthol (which I still largely employ), thymol, boracic acid, creolin, &c., &c., I have witnessed truly marvellous results in the treatment of phthisis. If I am asked why I have not brought these cases more frequently before the profession, my answer is—it is by no means easy for a general practitioner to gain even a hearing, as I could readily show.

Five years since, Dr. Sheridan Delépine's report on the bactericidal power of izal in destroying typhoid and tubercle bacilli caused me to try izal injected through the larynx in the treatment of pulmonary tuberculosis. I commenced with very small doses of the pure oil, which was not then on the market, but was supplied by special request, by the manufacturers. Assured of its non-toxic character (S. Delépine, *loc. cit.*, and "The Pharmacology of Izal," F. W. Tunnicliffe, *Pharmaceutical Journal*, April 18th, 1896), the dose was slowly increased—*guttatim*—until the maximum was reached, and I had proved it easily practicable to send down the windpipe 60 minims (3·5 c.c.) suspended in glycerine. I found it practicable to employ a 10 per cent. solution, and as it had been previously shown that a dilution of $\frac{1}{2}$ per cent. would destroy the bacillus coli and the staphylococcus pyogenes in five minutes, it appeared to me that—theoretically—the cure and prevention of tubercular phthisis had been already achieved. Three years ago I could have, clinically and microscopically, shown patients who had recovered, and sputum, once full of bacilli, from which all trace had disappeared; but, as I have before stated, I could not attract any attention. It was, perhaps, as well, for it is very difficult to prove to a scientific occasional observer that any case has either been cured, or that a cure is in progress. The more logically scientific the observer is, the more doubtful he would have been. We have been too often

Digitized by Google

hoaxed by consumption cures, even though they come from scientific Germany.

In October, 1899, I had the pleasure of an interview with Professor Sheridan Delépine, and asked if he could complete the experiments he had begun in 1895, to decide what strength of izal solution would be necessary to kill or inhibit the growth of tubercle bacilli in tuberculous sputum. This he most kindly promised to do, and his results were in my hands so long ago as May, 1900, and with his very kind permission I place the following quotations from this very important report before the Congress :—

"REPORT ON THE DISINFECTING PROPERTIES OF IZAL.

"SECOND COMMUNICATION: EXPERIMENTAL INVESTIGATION ON THE VALUE OF IZAL IN THE DISINFECTION OF TUBERCULOUS PRODUCTS AND IN THE TREATMENT OF TUBERCULOUS LESIONS.

"BY SHERIDAN DELÉPINE (*Proctor Professor of Pathology, Owens College*),

AND

"F. J. H. COUTTS, M.D. (*Assistant to the Professor of Pathology in the Bacteriological Department, Owens College*).

"In the first investigation of izal, published nearly five years ago, one of us * had stated, on the basis of a number of experiments, 'that izal mixed with ten parts of water will disinfect in forty-five minutes dried tuberculous sputum or other tuberculous matter, and that fresh tuberculous products of great virulence, when mixed with an equal quantity of izal of the same strength as above, and allowed to dry at the ordinary temperature for twelve hours, are also completely disinfected.' The minimum amount of izal sufficient to obtain these results had not, however, been ascertained; it was therefore necessary to complete this first investigation. This became all the more necessary that, as a result of clinical observations made by Dr. Colin Campbell, some hope had been entertained that izal might prove very useful in the treatment of pulmonary phthisis. The clinical effects of the administration of izal were not within the scope of laboratory experiments, but it was a matter of great interest and practical importance to ascertain whether it was possible to kill tubercle bacilli in the midst of living tissues, or at any rate to produce such changes in the tissues as to make them capable of resisting the tuberculous invasions.

"It was therefore with the double object—first, of testing further the value of izal as a bactericidal agent outside the body, and second, of finding out

* S. Delépine: "Report on the Disinfecting and Antiseptic Properties of Izal." 'Med. Chronicle.' Sept., 1895; also Klein: "Experiments in Izal," 1892.

Digitized by Google

whether its use as a curative agent was likely to be beneficial—that the present investigation was undertaken.

"1. Disinfection of Tuberculous Sputum by Izal.*

"The 'izal' used in these experiments was supplied to us by Mr. J. H. Worrall, the discoverer of the substance, who informed us that 'izal' is an emulsion prepared for medicinal and surgical use, and contains 40 per cent. of izal oil.

"By mixing 1 part of the emulsion with 9, 19, 49, and 99 parts of water respectively, various dilutions of the emulsion were obtained. The following table gives the amount of 'izal oil' contained in 1 c.c. of each of these dilutions:—

1 c.c. of $\frac{1}{10}$ dilution of the medicinal izal contains $\frac{1}{25}$ c.c. of pure 'izal oil.'
" $\frac{1}{25}$ " " " " $\frac{1}{50}$ " " "
" $\frac{1}{50}$ " " " " $\frac{1}{125}$ " " "
" $\frac{1}{100}$ " " " " $\frac{1}{250}$ " " "

"These solutions were mixed with sputum teeming with tubercle bacilli, so as to obtain mixtures containing definite proportions of izal. The following mixtures, in which the sputum and izal were thoroughly stirred together, were used in our experiments.

Quantity of Sputum.	Quantity of Emulsion.	Dilution of Emulsion.	Quantity of Izal Oil mixed with 1 c.c. of Sputum.
A.—5 c.c.	5 c.c.	$\frac{1}{10}$	$\frac{1}{25}$ c.c.
B.—8 c.c.	4 c.c.	$\frac{1}{10}$	$\frac{1}{50}$ c.c.
C.—8 c.c.	2 c.c.	$\frac{1}{10}$	$\frac{1}{100}$ c.c.
D.—5 c.c.	5 c.c.	$\frac{1}{20}$	$\frac{1}{50}$ c.c.
E.—5 c.c.	5 c.c.	$\frac{1}{20}$	$\frac{1}{125}$ c.c.
F.—5 c.c.	5 c.c.	$\frac{1}{100}$	$\frac{1}{250}$ c.c.

"With these various mixtures twelve guinea-pigs were inoculated. The

* From a comparison of the boiling point, specific gravity and ultimate analyses of the "izal oil" and of phenol, cresol and xylenol, Mr. Worrall has come to the conclusion that izal oil contains more hydrogen, in proportion to the carbon, than the corresponding member of the carbolic series; and that this substance consists of chemically oxidised hydrocarbons, having a larger proportion of hydrogen to carbon than the members of the $C_6 H_6 O$ series, and a less proportion than the members of the $C H_4 O$ series. It seems probable that izal oil is not a pure substance, but a mixture, which is insoluble in water and non-volatile at the ordinary temperature.

Digitized by Google

results of these experiments are given in detail in the report, from which the following summary has been prepared by Delépine :—

No of Experiments in each case.	Quantity of tuberculous sputum.	Quantity of pure izal oil added to the sputum.	Time allowed between the mixing of the izal and sputum and the inoculation.	Result of the inoculation.
2	1 c.c.	$\frac{1}{25}$ c.c.	1 hour	No tuberculosis.
4	1 c.c.	$\frac{1}{50}$ c.c.	1 hour	No tuberculosis.
1*	1 c.c.	$\frac{1}{100}$ c.c.	1 hour	No tuberculosis.
2	1 c.c.	$\frac{1}{125}$ c.c.	1 hour	No tuberculosis.
2	1 c.c.	$\frac{1}{250}$ c.c.	1 hour	Tuberculosis typical, but developing slowly.
2	1 c.c.	No izal	Controls	Typical tuberculosis.
Total .. 13				

"It is clear from these results that tuberculous sputum can be disinfected in one hour by being thoroughly mixed with an emulsion of izal containing $\frac{1}{125}$ c.c. of izal oil to each 1 c.c. of sputum.

* * * * *

"It seems therefore that izal can be relied upon for disinfecting tuberculous sputum, provided the proportion of pure izal oil to the sputum be as 1 to 125. For practical purposes it is evidently desirable, in order *to obtain thorough mixture of the disinfectant with the sputum*, to use a quantity of izal emulsion about equal to that of the sputum."

I do not intend to refer here to the second part of Professor Delépine's report.

I have had the privilege of demonstrating, this morning, the facility with which fluids can be injected through the larynx into the lungs. I desire to thank Dr. Hector Mackenzie for giving me the opportunity of doing so, and so kindly allowing four of his patients from the Brompton Hospital to attend here for this purpose. The patients were wholly unknown to me. I saw them for the first time yesterday afternoon, and I think it says something for the method that they were willing to continue to be injected as long as there was an audience to observe it. But with new and unknown patients the process was not ideal. My own patients would have easily taken twice or three times the quantity, two or three ounces, in one-third the time, and without a cough or least discomfort.

Surely it is more reasonable to send this potent bactericidal substance

* One of the two experiments in which $\frac{1}{100}$ c.c. of izal oil was used is not recorded, because the guinea-pig died accidentally six days after inoculation. All the other guinea-pigs remained *apparently* well, and were killed fifty-two days after inoculation.

Digitized by Google

into the part affected than to trouble the patient's stomach, which has already enough to do! May I quote what I said on this subject seven years ago? "To bring the remedial agent into direct contact with the diseased part is a principle so well recognised in surgery that it should require no argument to prove the rationale of extending its application to diseases of the respiratory system. Can anything be more faulty than our present system of attempting to render aseptic a lung cavity, filled with tenacious muco-pus, by a vapour, not a tenth part of which probably ever reaches the affected parts? . . . Who would attempt the treatment of a septic abscess by a vapour, if he could wash it out first and then send in his antiseptic solution, or fill it with aseptic absorbent? We are not yet in a position to attempt, with much hope of success, the washing out of a pulmonary cavity, but instead of the vapour, or smell, of an antiseptic, we can send in the antiseptic itself." (Campbell: Trans. Roy. Med. Chirurg. Society, vol. 78.) But if we cannot easily and safely wash out a pulmonary cavity, we can supply the lungs with something with which it washes itself out. In the solutions which I employ, glycerine is probably the most active agent in causing this. After a dose has been introduced, I direct patients to take deep inspirations. This is first followed by the sensation of heat produced by the fluid travelling through the lungs, and later by a greatly increased flow of expectoration. The treatment of chronic nasal catarrh by this method affords a capital object-lesson as to the superiority of the wash-out produced by an intra-nasal injection of one of these solutions, as compared with the ordinary douche. After a single injection of fifteen or twenty minims into each nostril, the nose will continue to discharge for several hours, and thick lumpy mucus will be discharged which a douche will have failed to dislodge.

Dosage-quantity of Fluid and Antiseptic possible to Inject.

Some years ago I pushed experiments to decide this, and found it was practicable to inject 3 oz. (90 c.c.) of fluid at a single sitting, and three doses of 2 oz. (60 c.c.) each in twelve hours. At that time I was using creosote, of which I found it possible to inject 6 drachms (22 c.c.) per day. In such doses, however, creosote produced decidedly unpleasant effects. It is, however, quite unnecessary to administer such quantities. The "bulk" I usually employ varies from 6 to 16 drachms (20–60 c.c.) at a sitting. When practicable it is best to give two doses a day, the first to wash out the lungs, the second to get to the bottom; but good results are obtained from a single dose daily.

According to Professor Delépine's experiments, izal diluted 125 times exterminates the tubercle bacillus in an equal quantity of sputum, if thoroughly mixed, and I have shown how the admixture is obtained—(1) by the dehydrating power of the solvent glycerine, and (2) by the action of respiration. Now suppose it be remotely possible that the lung or lungs of a living being should contain 1 pint (20 oz. or 568 c.c.) of muco-pus "teeming with

Digitized by Google

bacilli," 90 minims (5·3 c.c.) of izal should disinfect the whole, and this quantity it is easy to inject at a single sitting.

But I would not base my case on such an argument. In the first place I should regard a human being with a muco-pus capacity of 20 oz. as having lost too much lung tissue to render cure possible; if his lungs were healed he would want an outfit of gills. Second, because I do not pin my faith on a single effort at destruction; I rely rather on the gradual effacement of the bacilli, and would prefer smaller doses often repeated. It is best to mix our "disinfectant," for in association with the tubercle bacilli are usually found many other organisms. Again, by itself, izal is distinctly nasty, and just as we make stomachic medicines agreeable, so it is well to mix our intra-tracheal injections with menthol, thymol, or the like. And here again crops up the utility of glycerine. Its high fattening power abolishes the necessity of that putrescence of the past—cod-liver oil.

Gentlemen, I have troubled you with no cases and no statistics; either would be impossible to prove. I rely on the logical strength of the case I have placed before you. As to the merits or demerits of the latest fad—the open-air treatment—I do not further propose to enter. Not that I would for a moment deny the absolute necessity of plenty of fresh air, but alone, I assert, it is not sufficient. If the presence of the tubercle bacillus be admitted to be the cause of tubercular phthisis, surely its destruction *in situ* should be the first object to secure recovery. And if, as all authorities, including Dr. Koch, believe, the expectoration of living germs constitutes the most potent cause of the spread of the disease, surely the destruction of the germ, before expectoration, is the most reasonable method of checking its spread.

Thus, simultaneously, can cure and prevention be accomplished.

The CHAIRMAN (Sir J. W. MOORE): We are much indebted to Mr. Colin Campbell. Years ago he showed us very distinctly that a large quantity of fluid could be injected through the trachea without much harm, which very few people, who had not seen it done, would believe. I myself saw it done, and done with a fair amount of success. As to whether it always goes to the right place beyond the trachea is a different thing, but still it is a great fact to show that.

Digitized by Google

THE THEORY OF THE ACTION OF IGAZOL.

By Professor VINCENZO CERVELLO, *Palermo.*

Clinical experience having shown the good effects that igazol, which is a combination of formic aldehyde with chloral, terpene, and iodoform, produces in pulmonary consumption, this remedy should be more thoroughly studied, in order to discover its methods of working. With reference to this subject, I have made various experiments on both human beings and animals, with the following results :—

When the subject to be experimented on comes first under the influence of igazol, respiration becomes rather slower, then it becomes normal, or nearly so. The diminution is not sustained.

An examination of the respiratory chart shows that the volume of inspiration and expiration increases considerably. The graphic form of the respiratory movements does not change.

The centre of respiration in the first stages is subjected to an exciting influence, which, however, later on acts as a sedative.

The blood pressure remains unaffected.

Animals introduced into an igazolated atmosphere absorb more oxygen out of the air, and exhale more carbonic acid, than in a normal atmosphere.

Blood, under the influence of igazol, becomes of a brighter red hue, and remains so for a long period. This coloration is due to the more active oxydation of the hæmoglobin. In a microscopical examination the two bands of absorption of oxy-hæmoglobin may be observed.

Igazolated blood resists putrefaction for several days.

The red globules retain their shape and histological character longer than normal blood globules.

Blood in an igazolated atmosphere absorbs more oxygen, and gives off more carbonic acid, than in a normal atmosphere.

The muscular tissue and the organic tissues exhibit the same modification in the altered atmosphere.

These results show that igazol exercises a very powerful influence over the animal organism, but on the more important subject of its methods of action I hope to arrive at more definite conclusions when I publish the second part of my work, which is not yet completed.

Digitized by Google

LE TRAITEMENT DE LA TUBERCULOSE PAR L'IGAZOL.

PAR M. LE PROFESSEUR CERVELLO (*Palermo*).

LORSQUE je publiais les premières expériences cliniques sur les inhalations d'igazol dans le traitement de la tuberculose pulmonaire, j'affirmais qu'on pouvait en obtenir certainement de bons résultats à condition qu'il y eut le concours de tous les facteurs hygiéniques auxquels la réparation organique est confiée, et j'ai conclu en disant que je ne présentais pas à la thérapie un spécifique infallible, mais seulement un système de traitement local qui ne peut devenir utile que si toutes les autres conditions qui rendent l'organisme capable de la plus active résistance sont assurées.

En effet, ma méthode se propose le but d'aider ces énergies naturelles qui tendent à la guérison spontanée de la tuberculose pulmonaire.

Les expériences faites depuis environ trois années sur ma méthode, quand elle a été employée dans les conditions que j'ai établies, ont demontré que mes conclusions étaient exactes, bien plus je peux dire que les autres observateurs ont obtenu des résultats encore plus encourageants que les miens.

Dès le commencement de mon étude clinique j'essayais de me rendre compte du genre d'action de l'igazol, et j'annonçais, comme une simple hypothèse, qu'il s'agissait probablement d'une action oxydante, exercée par l'aldehyde formique sur les bacilles de la tuberculose et sur leurs produits, et que la nutrition générale et l'hématose devaient être aussi excitées. Selon ce mécanisme l'igazol réussirait à imiter fidèlement la nature.

Pour appuyer cette hypothèse je n'avais pas de données expérimentales, que j'ai seulement pu obtenir dans ces derniers temps et que j'ai l'honneur de communiquer à cette noble assemblée.

Dans mes expériences j'ai employé l'igazol, c'est-à-dire l'association de l'aldehyde formique au chloral hydrate à la terpine et au iodoforme, parce que mon but était justement de connaître l'action de cette association. Je ferai connaître, lorsque j'aurai fini toutes les expériences que je suis en train de faire, quel est le rôle que joue chacun de ces corps.

L'influence que les inhalations d'igazol exercent sur la nutrition générale est rapide et considérable. Je compris bientôt qu'elle ne peut pas être seulement une conséquence de l'atténuation des symptômes de la tuberculose pulmonaire, parce que même les individus sains qui faisaient les inhalations et ceux qui travaillaient à la préparation du médicament montraient les mêmes effets. J'en concluais donc que l'igazol n'agissait pas seulement sur les phénomènes morbides, mais qu'il devait exciter directement les fonctions de nutrition générale.

J'ai fait des expériences sur des animaux sains : des rats blancs et des lapins, auxquels je donnais à manger à satiété. Les animaux étaient pesés tous les jours, et quand le poids restait constant je leur faisais faire des

Digitized by Google

inhalations d'igazol, pendant trois heures par jour, en préparant l'atmosphère avec les mêmes proportions, comme on procède pour l'homme.

Voici les résultats obtenus :—

RATS BLANCS.

Jour.	I. Poids du corps.	II. Poids du corps.	III. Poids du corps.
	gr.	gr.	gr.
7 juin	142	107	137
28 juin	153	131	154

LAPINS.

Jour.	I. Poids du corps.	II. Poids du corps.	III. Poids du corps.	IV. Poids du corps.
	gr.	gr.	gr.	gr.
28 juin. . .	1,950	1,731	1,510	2,070
2 juillet . .	1,952	1,750	1,550	2,150
5 ,, . .	2,020	1,820	1,625	2,200
8 ,, . .	2,050	1,870	1,760	2,260
10 ,, . .	2,060	1,871	1,760	2,260
12 ,, . .	2,062	1,869	1,761	2,261

L'augmentation du poids est donc considérable, elle s'obtient rapidement, c'est-a-dire après dix jours environ.

En calculant par kg. d'animal on trouve dans les rats une augmentation maximum de gr. 110,38 par kg. et dans les lapins de gr. 142,50 par kg.

En même temps on remarque une augmentation dans le chiffre de l'hémoglobine, laquelle, mesurée avec l'hémomètre de Fleischl, donne une différence en plus de 7 dans le lapin I., de 10 dans les lapins II. et IV., de 8 dans le lapin III.

Les fonctions respiratoires subissent des modifications importantes. J'ai fait des expériences sur l'homme et sur des chiens.

On mesure le fréquence respiratoire d'abord dans les conditions normales et ensuite pendant le séjour dans l'atmosphère igazolée. Les résultats obtenus montrent que la fréquence respiratoire subit au commencement une légère diminution, de cinq respirations au maximum, et après une augmentation qui atteint quelquefois le chiffre normal. Parfois cette diminution n'a pas lieu, ou bien elle est très faible.

Si au lieu de faire habituer graduellement l'individu à l'atmosphère igazolée, comme on a procédé précédemment, on le fait entrer subitement dans cette atmosphère, on obtient presque les même résultats.

Digitized by Google

Les modifications des excursions respiratoires sont encore plus importantes.

La fig. I. représente la courbe respiratoire prise sur l'homme avant les inhalations, et la fig. II. celle prise pendant les inhalations.

Les fig. III. et IV. sont prises sur le chien dans les mêmes conditions.

On aperçoit de suite que les excursions du torax se font aussi plus larges, deux à quatre fois plus grandes que les normales. Cette plus grande dilatation persiste pendant toute la durée des inhalations.

En même temps que les modifications mentionnées on obtient aussi une action sédative sur la fonction respiratoire. Celà est demontré en mesurant le temps pendant lequel un individu ayant le nez et la bouche fermés peut rester sans respirer. Ce temps, après les premières quinze minutes, diminue de cinq à huit secondes, mais après commence à augmenter jusqu'à atteindre le normal et même le surpasser de dix secondes.

Il résulte de ces expériences que l'igazol diminue un peu la fréquence respiratoire, fait augmenter sensiblement l'amplitude du torax et agit, en outre, comme sédatif de l'innervation de la fonction respiratoire. Ces modifications nous expliquent le soulagement que les phthisiques éprouvent pendant les inhalations. Ils accomplissent une véritable gymnastique pulmonaire, tandis que la toux, aussi bien que l'accélération respiratoire, viennent s'apaiser.

On sait par les expériences faites d'abord par Vierordt et G. Ludwig, et ensuite par Marey, que l'amplitude de la courbe respiratoire est proportionnelle au volume de l'air mise en mouvement par la respiration, c'est-à-dire que les mouvements externes de la respiration ont une intensité proportionnelle à la quantité d'air que le torax aspire et expire. Nous pouvons donc en conclure que les inhalations d'igazol augmentent la ventilation pulmonaire.

Ce résultat n'est pas sans intérêt dans le traitement de la tuberculose pulmonaire.

Maintenant il s'agit de connaître si, à cette plus grande introduction d'air, correspond une plus grande fixation d'oxygène. A ce sujet j'ai fait les expériences suivantes.

Un rat blanc est introduit dans une cloche hermétiquement close par un bouchon de caoutchouc, traversé par un tube de verre qui porte à son extrémité un tube de caoutchouc fermé par une pince. La cloche a une capacité de 10312 cc., de sorte que l'animal peut y demeurer longtemps sans éprouver aucune souffrance.

Après un séjour de deux à trois heures on met la cloche en communication avec l'appareil de Hempel, et on mesure le pourcentage d'oxygène et d'anhydride carbonique. Ensuite on porte la cloche, après en avoir renouvelé l'air, dans la chambre préparée avec les vapeurs d'igazol qu'on fait pénétrer en insufflant au moyen d'un soufflet, l'air de la chambre, de sorte que dans la cloche l'atmosphère est igazolée dans les mêmes proportions que celles de la chambre. Après celà le rat est de nouveau introduit dans la cloche pour y demeurer le même temps qu'auparavant. On fait alors une seconde déterminati on du pourcentage d'oxygène et d'anhydride carbonique dans la cloche.

L'expérience est donc faite de la manière la plus simple en reproduisant

Digitized by Google

à peu près les mêmes conditions dans lesquelles se trouve le phtisique qui est soumis à ma méthode.

Pour chaque expérience on mesure d'abord la proportion de l'oxygène de l'air atmosphérique de la chambre où l'on travaille. L'anhydride carbonique est en si faible quantité qu'on ne peut pas la doser.'

L'expérience disposée comme je viens de le dire permet de connaître la quantité d'oxygène consumée par le rat dans la première période et celle absorbée dans la seconde période pendant les inhalations.

Je transcris quelques expériences faites :—

1. Rat blanc de gr. 143.

Après un séjour de 2 h. 40 m. dans l'atmosphère normale on trouve dans la cloche 13,06 pour cent d'oxygène et 6,48 pour cent d'anhydride carbonique ; après le même séjour dans l'atmosphère igazolée on trouve 12,24 pour cent d'oxygène et 7,28 pour cent d'anhydride carbonique.

Le calcul fait en grammes par heure et par kg. d'animal donne les chiffres suivants :—

O fixé dans l'atmosphère normale	0,253 gr.
,, ,, igazolée	0,282 ,,
Diff.	0,029 gr.
CO_2 éliminée dans l'atmosphère normale	0,311 gr.
,, ,, igazolée...	0,355 ,,
Diff.	0,044 gr.

2. Le même Rat.

Après un séjour de trois heures dans l'atmosphère normale on trouve dans la cloche 12,53 pour cent d'oxygène et 6,69 pour cent d'anhydride carbonique ; après le même séjour dans l'atmosphère igazolée on trouve 11,52 pour cent d'oxygène et 8,98 pour cent d'anhydride carbonique.

Le calcul fait en grammes par heure et par kg. d'animal donne les chiffres suivants :—

O fixé dans l'atmosphère normale	0,272 gr.
,, ,, igazolée	0,308 ,,
Diff.	0,036 gr.
CO_2 eliminée dans l'atmosphère normale	0,326 gr.
,, ,, igazolée...	0,436 ,,
Diff.	0,110 gr.

En comparaison des phénomènes qui se produisent en respirant dans l'atmosphère normale on aperçoit qu'en respirant dans une atmosphère

Digitized by Google

igazolée une plus grande quantité d'oxygène est fixée et une plus grande partie d'anhydride carbonique est éliminée.

Sous l'influence de l'igazol, à la plus grande quantité d'air qui pénètre dans le torax, correspond donc une plus vive hématose.

Cette accélération de l'échange gazeux s'accomplit sous l'influence de l'igazol, non seulement dans le sang en circulation, mais encore dans le sang hors de l'organisme et dans les tissus organiques.

Cela est démontré par les expériences suivantes : On introduit dans deux flacons de capacité égale deux quantités égales de sang défibriné, ou de muscles, ou de morceaux de grenouilles ; les deux flacons sont bouchés. Le premier contient de l'air atmosphérique, l'autre des vapeurs d'igazol qu'on a fait pénétrer à l'aide d'un soufflet comme on a procédé pour la cloche. Après quelques heures on mesure, à l'aide de l'appareil de Hempel, le pourcentage d'oxygène et d'anhydride carbonique contenue dans chaque flacon.

De ces expériences résulte que par chaque litre de sang de bœuf défibriné, l'absorption d'oxygène pendant une heure dans l'atmosphère igazolée est plus grande de gr. 0,380 et l'élimination d'anhydride carbonique est plus grande de gr. 0,250.

Des morceaux de grenouilles donnent en plus gr. 0,810 pour l'oxygène et gr. 1,724 pour l'anhydride carbonique.

Je crois donc avoir démontré que l'igazol excite les tissus organiques à fixer et à consommer des quantités d'oxygène plus grandes que dans les conditions ordinaires. Je crois, par conséquent, pouvoir en conclure qu'il s'agit d'une action oxydante très énergique exercée par l'igazol.

Cette oxydation qui s'accomplit dans le sang n'altère pas tout-à-fait les caractères de l'hémoglobine. Quand on fait traverser le sang par des vapeurs d'igazol il acquiert une coloration rouge plus vive qu'il conserve même pendant quelques jours durant lesquels il résiste à la putréfaction.

Si le sang est veineux il devient bientôt artérieux.

Quand on examine au spectroscope le sang, même fortement igazolé, on remarque que les bandes d'absorption de l'hémoglobine sont inaltérées.

L'action de l'aldéhyde formique sur l'hémoglobine a été mise en relief par Tollens : lorsqu'on ajoute au sang qui contient l'aldéhyde formique du sulfure d'ammoniaque et qu'on chauffe légèrement, les deux bandes de l'hémoglobine vont graduellement disparaître, et il se forme entre elles une troisième bande très marquée, celle de l'hémoglobine réduite. Si on agite ensuite avec de l'air cette bande disparaît, et les deux premières réapparaissent.

Ces phénomènes se répètent sur le même sang toutes les fois qu'on recommence à le réchauffer ou à l'agiter.

Cela signifie, selon moi, que l'aldéhyde formique opère sur le sang successivement des réductions et des oxydations. Mais je ne m'arrête pas, pour le moment, sur ce sujet, auquel je reviendrai lorsque j'aurai fini mes expériences sur la propriété oxydante de l'aldéhyde formique, mais dès maintenant on comprend bien le rôle important que joue cette propriété oxydante dans le traitement de la phtisie pulmonaire.

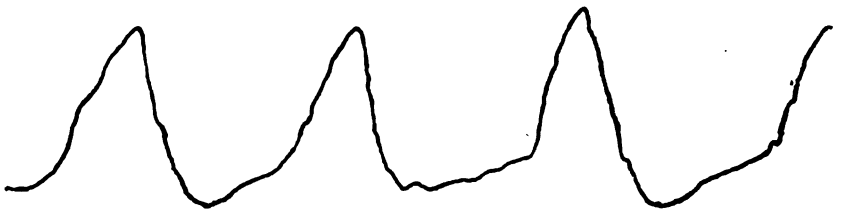
Digitized by Google

Bien que mes recherches ne soient pas encore terminées je crois cependant que si les faits, trouvés jusqu'à présent, n'arrivent pas à expliquer tout le mécanisme avec lequel agissent les inhalations d'igazol, ils montrent déjà la puissante action qu'elles jouent sur l'organisme animal.

FIG. I.

HOMME—COURBE RESPIRATOIRE NORMALE.

FIG. II.



HOMME—COURBE RESPIRATOIRE PENDANT LES INHALATIONS D'IGAZOL.

FIG. III.

CHIEN—COURBE RESPIRATOIRE NORMALE.

FIG. IV.

CHIEN—COURBE RESPIRATOIRE PENDANT LES INHALATIONS D'IGAZOL.

Digitized by Google

TREATMENT OF PULMONARY TUBERCULOSIS BY HYPODERMIC INJECTIONS OF PHOSPHATE OF LIME ($CaH_4[PO_4]_2H_2O$).

By Dr. GUSTAVE ANGIULLI,

Agrégé Volontaire in the Second Medical Clinic at Naples.

The hypodermic injection of phosphate of lime, which, so far as I am aware, has never been employed before in the treatment of pulmonary tuberculosis, is intended :—

(1) Powerfully to augment the general resistance of the diseased organism by supplying it, in a form capable of rapid and easy assimilation, with a mineral substance of the first importance in the body, existing as it does in every tissue and in all secretions. This substance is freely eliminated throughout the disease, and sometimes even before any illness has shown itself (pre-tubercular phosphaturia), and if its loss is not rapidly made good the organism is progressively demineralised, with grave results, and the general resistance of the body—so necessary in the successful struggle against tuberculosis—is seriously impaired.

(2) To act powerfully on the nervous system, which is early attacked by tubercular poisons (toxins). This attack is shown, I think, by the increased excretion of phosphate of lime in the urine, a symptom which may precede all pulmonary signs, and may therefore be spoken of as "pre-tubercular phosphaturia." I consider this to be a toxic phosphaturia, and the physician should be on the lookout for it in all suspected cases of pulmonary tuberculosis.

The injections have been very well borne by patients, and when carefully made have caused no serious inconvenience.

Digitized by Google

TRAITEMENT DE LA TUBERCULOSE PULMONAIRE PAR LES INJECTIONS HYPODERMIQUES DE PHOSPHATE DE CHAUX ($CaH_4[PhO_4]_2H_2O$).

VALEUR THÉRAPEUTIQUE DE LA PHOSPHATURIE PRÉTUBERCULAIRE, EXPLIQUÉE COMME PHÉNOMÈNE PRÉCOCE D'INTOXICATION DU SYSTÈME NERVEUX.

PAR M. LE DR. GUSTAVE ANGIULLI (*Naples, Italie*), *Agrégé Volontaire à la Deuxième Clinique Médicale de Naples.*

MESSIEURS,—Au Congrès contre la Tuberculose, tenu à Naples dans le mois d'avril de l'année passée, de même qu'au Congrès international à Paris, j'ai rapporté les heureux résultats obtenus sur quelques malades de Tuberculose pulmonaire, soignés par mes injections hypodermiques de phosphate acide de chaux. Après environ un an, je puis reconfirmer, ici, les grands advantages de cette cure, qui a pour but de fortifier, puissamment, l'organisme malade, en atténuant, et, s'il est possible, en arrêtant sa funeste déminéralisation progressive.

Vous connaissez bien, Messieurs, le rôle important que la chaux joue dans l'échange organique : combinée, surtout, avec l'acide phosphorique, elle entre dans tous les liquides, dans tous les solides de notre économie. Vous savez bien qu'elle se trouve dans toutes les cellules capables d'un développement, surtout dans leur nucléum, et qu'elle abonde spécialement dans la molécule nerveuse en général, et dans la molécule cérébrale en particulier. Il est donc facile de comprendre le dommage que doivent produire dans l'organisme les pertes considérables du phosphate de chaux, qui, dès l'abord, ont lieu dans la Tuberculose pulmonaire, si elles ne sont pas, bientôt, réparées d'une manière convenable. En effet, leur persistance déminéralise l'organisme et en diminue la résistance générale, si nécessaire à combattre la maladie.

Le but de mes injections hypodermiques a, donc, été, avant tout, d'atténuer, justement, ces pertes et de tâcher d'y réparer, de la façon la plus rapide, en choisissant, à cet effet, la voie la plus sûre pour une prompte assimilation. J'ai fait préparer, pour cela, des solutions rigoureusement stérilées de phosphate acide de chaux pour que le sel—tout en conservant parfaitement sa formule moléculaire—fût cependant privé de l'excès d'acide phosphorique, qui, à cause de son action locale irritante, l'aurait rendu peu propre à être injecté sous les tissus.

Mes expériences ont prouvé que le phosphate de chaux a, toujours, été bien assimilé par l'organisme malade. Sa quantité alla toujours diminuant dans les urines, en même temps que le poids du corps augmentait, et que

Digitized by Google

tous les symptômes de la maladie subissaient une amélioration fort remarquable, jusqu'à disparaître complètement dans les cas pas trop avancés. Et que l'on remarque bien que, pendant la cure, je n'ai voulu changer, en rien, ni l'alimentation, ni les conditions hygiéniques dans lesquelles le malade se trouvait auparavant, et cela pour pouvoir attribuer, exclusivement, à la cure l'amélioration que l'on pouvait en obtenir.

Les injections, pratiquées selon les règles antiseptiques les plus scrupuleuses, ont toujours été bien tolérées par les malades, et n'ont jamais donné lieu à aucun inconvénient sérieux.

Mais, après les observations faites et les résultats obtenus, et après m'être assuré que le phosphate de chaux avait toujours été—même dans les cas très avancés et très graves—bien assimilé et utilisé par l'organisme malade, j'ai, depuis peu, réfléchi aux avantages inestimables qu'il pourrait procurer, si, au lieu de combattre le mal, déjà déclaré, on l'employait à combattre et à arrêter un symptôme de la plus grande importance, expression initiale d'un commencement de Tuberculose. J'entends parler de la phosphaturie prétuberculaire qui se trouve, comme l'albuminurie, dans les urines des phthisiques, même avant que toute autre manifestation de la maladie soit appréciable. A ce que je sache, il ne me semble pas qu'on ait donné à cette phosphaturie, déjà décrite par Teissier de Lyon, la signification clinique très importante qu'elle a en réalité, et qui est d'une immense valeur pour le diagnostic précoce du mal et, surtout, pour sa cure en temps opportun pour l'arrêter et le vaincre.

Je crois, en vérité, que cette phosphaturie prétuberculaire doit être expliquée comme la résultante de cette action toxique, complexe et continue, que les toxines tuberculaires exercent sur le système nerveux. En effet, depuis l'instant où le bacille de la Tuberculose envahit le poumon et s'y établit, jusqu'au moment où la lésion, produite par lui, devient appréciable, il s'écoule un certain temps, pendant lequel aucun symptôme du mal n'est aperçu par le malade, et, moins encore, par le médecin.

Cependant le bacille vit et secrète ses produits toxiques, une partie desquels, circulant dans l'organisme, va expliquer son action nuisible sur le système nerveux. Il est bien naturel qu'à cette action excitante et perturbatrice que, dès les premiers moments, les produits toxiques exercent sur le système nerveux, celui-ci réponde par une réaction proportionnée, et, par conséquent, par un travail plus vif et une consommation plus grande de son énergie potentielle. L'expression de ce travail plus vif, dû à une cause nouvelle et anormale, est, justement, selon moi, la phosphaturie prétuberculaire. Cette conviction me vient de la physiologie et de la clinique : en effet, la première nous apprend que l'augmentation dans l'élimination de l'acide phosphorique est toujours due à une destruction plus considérable de la substance nerveuse et que le travail cérébral augmente, toujours, la quantité des phosphates dans les urines ; et, à son tour, la seconde confirme que, dans les maladies nerveuses en général, le phosphate de chaux augmente presque constamment.

Quelle que soit donc la cause, normale ou morbide, qui oblige le système nerveux à un travail supérieur au travail ordinaire, un des symptômes

Digitized by Google

principaux et des plus constants est l'augmentation des phosphates dans les urines, et, surtout, du phosphate de chaux.

La phosphaturie prétuberculaire ne serait donc pas, comme beaucoup de monde le croit, l'indice exclusif de la déminéralisation du poumon ; mais elle serait, en grande partie, l'effet de l'action toxique des poisons tuberculeux sur le système nerveux. Et, en vérité, nous ne saurions nous expliquer cet état de lassitude générale et de malaise que ressentent les malades, dès le commencement du mal, comme dépendant uniquement de la lésion locale. Au contraire, nous nous l'expliquons très bien en considérant cette lésion comme le foyer actif de production des principes toxiques qui empoisonnent, lentement, l'organisme, en agissant, de préférence, sur le système, de l'équilibre dynamique duquel dépend toute la résistance organique, toute notre vie.

Si cette action nuisible continue des substances bacillaires pouvait être paralysée, de façon telle que la déperdition majeure de l'énergie nerveuse, qu'elles produisent, pût être, au fur et à mesure, réparée par une assimilation de la substance, proportionnée à la quantité perdue, il n'y a point de doute que l'équilibre dynamique du système nerveux resterait inaltéré et que le ton de résistance générale qui, de lui, se répand dans tout l'organisme, ne serait pas, progressivement, détruit. Et alors les tissus, non encore dépourvus de l'influence protectrice des nerfs, se trouveraient en condition de résister davantage à l'infection, laquelle trouverait, en eux, un terrain certainement peu propice à son développement continu.

Malheureusement, cela n'arrive point dans la très grande majorité des cas. La tuberculose atteint les individus déjà affaiblis par des causes héréditaires ou acquises. Cette faiblesse retentit, naturellement, sur toutes les fonctions organiques, spécialement sur les fonctions gastro-entériques, de sorte qu'il n'y a pas à espérer une assimilation nutritive normale et active. Le travail excessif, auquel est soumis le système nerveux, peut, donc, bien difficilement être réparé par une nutrition proportionnée et par une assimilation bonne et complète du matériel détruit.

Et cela, en partie, parce que, dans cette première période, ni le malade ni le médecin se sont encore aperçus du danger imminent. Par conséquent, l'action bienfaisante et protectrice du système nerveux s'affaiblit par degrés, entraînant une diminution de cette résistance naturelle et organique, qui a, dans ce système, son régulateur souverain et suprême. Aux causes de la faiblesse organique originaire, héréditaires ou acquises, il s'en adjoint donc, et dès les premiers temps de la maladie, une autre, qui n'est certes ni moins grave, ni moins à craindre que les autres.

Il résulte de tout ce qui précède, qu'il serait, selon moi, de la plus grande importance clinique et thérapeutique de chercher à temps la phosphaturie prétuberculaire qu'il serait mieux, comme je pense, dire "tuberculaire," parce qu'elle n'est qu'une manifestation précoce de la maladie.

C'est elle qui apprend au médecin qu'il se passe quelque chose de nuisible pour le système nerveux, et, par conséquent, pour l'organisme entier. Eh bien, si, après un examen consciencieux, il ne peut rapporter ce symptôme à une affection morbide bien précisée ; il peut le retenir, avec une grande

Digitized by Google

probabilité, comme l'expression sûre d'une tuberculose, à sa phase initiale. Et cette probabilité deviendra presque de la certitude, s'il a affaire à des individus qui, soit par des raisons héréditaires, soit à cause d'autres maladies infectives et épuisantes, soit à cause de graves excès et souffrances morales, sont dans les conditions les plus propices pour contracter ce mal terrible.

Je crois, Messieurs, que voilà le meilleur moment pour un traitement thérapeutique actif, énergique et rationnel. Le mal est à son premier commencement; l'organisme n'est pas encore profondément empoisonné par les ptomaines microbiques; la résistance n'est, pas encore, sérieusement compromise, et notre intervention thérapeutique, opportune et rationnelle, pourra presque certainement remporter la victoire. Nous savons fort bien que ce n'est pas le microbe qui fait la maladie; mais bien le pouvoir de réaction de l'organisme contre le parasite envahisseur. Ne laissons donc pas passer, les bras croisés, cet instant précieux, en lui laissant le chemin libre. C'est le vrai moment d'une action curative, capable de sauver le malade; plus tard, nous ne pourrons peut-être, dans la majorité des cas, qu'assister au progrès continu du mal, impuissants à l'arrêter malgré notre immense et—hélas!—presqu'inutile arsenal thérapeutique.

Mes nouvelles observations, faites selon ces idées et ayant pour but de combattre le mal à son premier début, sont commencées il y a trop peu de temps pour que je puisse aujourd'hui en rapporter des résultats complets.

Il me suffit, pour le moment, d'avoir appelé l'attention de mes confrères sur ce symptôme fort important de la phosphaturie tuberculaire, parce qu'il nous permet de reconnaître et de soigner à temps le mal, qui, latent en apparence, mine déjà, lentement, mais continûment, l'existence du malade. La phosphaturie tuberculaire est facile à trouver, pourvu qu'on ait le soin de la chercher.

Il suffit, en effet, que le médecin, soigneux et prévoyant—dans tous les cas où il peut, même de loin, supposer l'existence du mal—ait recours à l'examen scrupuleux des urines, le répétant, s'il le faut, plusieurs fois de suite. En agissant de la sorte, s'il trouve une augmentation remarquable de phosphate de chaux, sans pouvoir en attribuer la présence à une autre cause, il aura ce qui lui suffit pour le diagnostic précoce du mal, et il comprendra quels devoirs impérieux, quelle responsabilité suprême lui incombent. Il est inutile de dire que dans les cas où tout doute diagnostique n'a point disparu, on pourra avoir recours à la preuve clinique de la tuberculine. Pour ma part, aussitôt que j'aurai vu un symptôme pareil, j'aurai immédiatement recours aux injections hypodermiques de phosphate acide de chaux, en proportionnant bien, selon les cas et autant qu'il est possible, la quantité du liquide à injecter. J'y unirai, ainsi que je l'ai déjà fait, l'usage de la viande crue et du plasma musculaire, selon la méthode Richet-Héricourt. Et cela, pour ajouter, à l'action réparatrice et réconstituante du phosphate acide de chaux, l'action si puissamment antitoxique de la viande crue et du plasma musculaire.

Je continuerai ainsi, avec une constance tenace, la lutte contre ce mal terrible, convaincu, avec Pasteur, "qu'en fait de bien à répandre, le devoir ne cesse que là où manque le pouvoir de faire plus et mieux."

Digitized by Google

(IIème COMMUNICATION.) TRAITEMENT DE LA TUBERCULOSE PULMONAIRE PAR LES INJECTIONS DE PHOSPHATE DE CHAUX ET LA VIANDE CRUE OU LE PLASMA MUSCULAIRE.

MESSIEURS,—En unissant à mes injections hypodermiques de phosphate acide de chaux l'usage de la viande crue et du suc musculaire, selon la méthode Richet-Héricourt, j'ai voulu, ainsi que je l'ai déjà dit, atteindre le double but d'avoir, en même temps, l'effet réconstituant du phosphate de chaux, et l'effet puissamment antitoxique de la viande crue et du plasma musculaire, que les illustres professeurs français avaient vu se produire dans leurs nombreuses et très importantes observations expérimentales et cliniques.

Et cela parce que, dans les cas graves, soignés seulement par mes injections hypodermiques de phosphate de chaux, j'avais bien remarqué une diminution positive de tous les symptômes, mais j'avais, aussi, constaté que la fièvre s'y était montrée fort résistante à la cure; et que, dans les cas très graves, ce symptôme principal de l'intoxication tuberculaire, quoique un peu diminué n'avait jamais, disparu complètement. Les malades que, pendant dix mois, j'ai soignés par cette méthode mixte, ont été au nombre de sept. Dans ce nombre, il y en a un qui avait une tuberculose galopante; trois, une tuberculose chronique à la deuxième phase; trois, une tuberculose chronique entre la première et la deuxième phase. Quatre avaient des précédents héréditaires; les trois autres n'avaient que des données de maladies affaiblissantes communes. Je fus appelé auprès du malade de tuberculose aiguë lorsque, déjà depuis environ trois mois, il était en proie à une fièvre quotidienne très élevée, de 40 degrés et même davantage, à type continu, avec de légères rémissions et accompagnée de sueurs extrêmement abondantes. Il suffit de dire que pour lui enlever ses chemises, on était obligé de les couper, tant elles adhéraient à son corps. Le malade était dans un état général de profond épuisement. Je commençai la cure mixte, en lui faisant, d'abord, prendre le suc musculaire extrait de la pression de 500 grammes de viande; puis, petit à petit, je portai cette quantité à deux kilogrammes et demi. Le suc fut fort bien toléré par le malade. Les sueurs commencèrent peu à peu à diminuer, jusqu'à disparaître tout-à-fait après un laps de dix jours. Il est inutile de faire ressortir toute la grande importance de la disparition de l'un des principaux phénomènes toxiques de la tuberculose. Elle frappe d'elle-même, si l'on considère qu'aucun des remèdes spécifiques communs ne fut employé pour atteindre ce but. En même temps, la fièvre diminua aussi, en oscillant entre 38 et 39 degrés, mais elle ne cessa jamais complètement: l'état général s'améliora, de même que la toux et les fonctions gastro-entériques. Mais, malgré tout cela, après environ deux mois de cure, le malade succomba, tout-à-coup, à une paralysie cardiaque. Il est certain que si la cure eût été commencée plus tôt, les résultats auraient été bien différents. Et j'exprime cette certitude parce que, malgré que les conditions du patient fussent excessivement graves au début de la cure, il y eut, pourtant, une amélioration

Digitized by Google

remarquable. La disparition complète des sueurs, la diminution de la fièvre, d'abord si élevée, m'ont tout-à-fait convaincu de la réelle et puissante valeur antitoxique du plasma musculaire.

Des autres cas, il y en a deux qui, depuis plusieurs mois, ne présentent plus aucun symptôme du mal. Les deux malades avaient, avant la cure, de la toux, de la fièvre et des sueurs, et l'un deux, une jeune femme, la suppression complète de ses règles. Tous ces symptômes ont disparu, depuis plusieurs mois, et jusqu'à ce jour, les deux sujets jouissent d'une bonne santé. Vu les conditions gastro-entériques passables, la cure fut faite par la viande crue, qui fut toujours facilement prise et bien tolérée. La quantité journalière fut de 300 à 500 grammes. Des deux malades, l'un a abandonné la cure depuis quelques mois ; l'autre la continue encore, après environ un an.

Les trois autres patients ont eu tous une amélioration bien remarquable : cessation de la fièvre et des sueurs ; disparition presque complète de la toux et de l'expectoration ; retour complet des forces au point de leur permettre de sortir de chez eux et de vaquer, en grande partie, à leurs affaires, ce qui leur était auparavant absolument impossible. Cependant les lésions locales, quoique de beaucoup améliorées, n'ont pas encore complètement disparu. Le traitement dure, pour deux malades, depuis environ neuf mois ; pour le troisième, depuis environ quatre mois. La quantité de viande crue ingérée a été de 400 à 600 grammes, parce qu'il n'a pas été possible d'en faire tolérer davantage. De temps à autre, il leur a été administré du suc musculaire en même temps que la viande crue, dont la quantité a été alors diminuée.

Le dernier cas se rapporte à un enfant de sept ans, sans précédents héréditaires, atteint d'une broncho-alvéolite et d'une pleurésie tuberculaire. La cure ne date que d'un mois environ. Elle a été commencée quand la maladie durait déjà depuis six mois, et avait résisté à tout traitement.

Jusqu'à ce jour, il n'y a eu aucune amélioration sensible, si ce n'est une diminution dans les sueurs.

De l'ensemble de ces cas et des résultats obtenus par la cure j'ai puisé la ferme conviction que la viande crue et le plasma musculaire ont une valeur antitoxique réelle et fort remarquable, et que, dans la tuberculose pulmonaire, ils peuvent constituer un vrai remède héroïque, pourvu qu'il soit bien appliqué et en temps utile, et non pas lorsque le mal a déjà produit des destructions telles et des phénomènes d'intoxication organique si graves qu'aucun remède humain ne peut plus sauver le malade.

Digitized by Google

DU CACODYLATE DE GAÏACOL DANS LE TRAITEMENT DE LA TUBERCULOSE.

PAR M. LE DR. F. BARBARY, *Nice.*

LE cacodylate de gaïacol, obtenu pour la première fois par nous et Monsieur Rebec, est utilisé depuis 1899 dans le traitement de la tuberculose.

Produit chimiquement défini $AS(CH^3)^2O^2-(C^6H^4-OCH^3)$, nous l'employons en injection et dissous dans une préparation huileuse représentant pour une ampoule de dix grammes d'huile stérilisée 0·0354 d'acide cacodylique correspondant à 0·05 de cacodylate de soude et 0·05 de gaïacol.

Nous injectons une ampoule tous les deux jours. Le traitement est interrompu pendant huit jours et repris.

Le cacodylate de gaïacol agit :

1° Sur la nutrition générale en renforçant l'organisme par l'acide cacodylique ;

2° Sur la tuberculose elle-même, comme modificateur du terrain et comme aboutissant à l'évolution arthritique par le gaïacol.

Nous avons pu établir des comparaisons avec les traitements habituels sur 50 malades. Le traitement par le cacodylate de gaïacol, joint à l'hygiène antituberculeuse, nous a donné, à la première et à la seconde périodes de la maladie, des résultats rapides et durables.

TUBERCULOSE ET SÉRUMS ANTITOXIQUES.

PAR M. LE DR. BARADAT, *de Cannes.*

DEPUIS que Richet et Héricourt, Bertin et Pick en 1889, Boinet en 1894, Maragliano, Behring et Knor en 1895, ont demontré les propriétés immunisantes, pour la tuberculose, du sang et du sérum d'animaux réfractaires aux maladies microbiennes, les recherches se sont multipliées et les applications de la sérothérapie à la tuberculose sont devenues de plus en plus nombreuses.

C'est, dit le Professeur Landouzy, l'idée de secourir " les néo-tuberculeux, à l'heure des premières atteintes et des premières défaillances," alors que les poisons des associations microbiennes ne les ont pas envahis, qui a engagé les médecins à utiliser " les moyens immunisateurs antitoxiques ou microbicitants par renforcement des activités phagocytaires."

L'emploi du sérum et des toxines anti-tuberculeuses dans le traitement de la tuberculose est-il vraiment efficace ? Nous répondons oui.

En effet, les observations montrent que tout sérum est un dynamogène et par conséquent un réconstituant général.

Digitized by Google

" Nous en voyons la preuve tous les jours dans l'emploi des sérums artificiels, dans les cas d'hémorragies graves, d'anémie consécutive aux maladies chroniques, dans les chocs traumatiques opératoires. Ce qu'on peut dire sur la valeur des sérums artificiels, comparée à celle des sérums naturels, dans la tuberculose, et c'est un point de pratique sur lequel j'insiste, c'est qu'une petite quantité de sérum naturel est nécessaire pour produire un effet thérapeutique intense, effet qui ne peut être obtenu que par une quantité double ou triple de sérum artificiel. Il y a là un *quid divinum* dû évidemment à la composition intime du sérum naturel. Personne ne nie plus l'action dynamogène des sérums, elle est reconnue."

" Il suffira donc de la repeter pour entretenir le sujet dans un état de résistance remarquable."

" En attendant que le laboratoire ou l'expérimentation aient éclairé cette question encore obscure du mode d'action des sérums, nous nous rattachons à la theorie de Metschnikoff, qui fait des sérums non pas des antitoxiques mais des stimulants des cellules phagocytaires autrement dit des stimulines, des provocateurs de résistance organique."

J'ajouterai que le professeur Ferran, de Barcelone, dont l'autorité scientifique est universellement reconnue, nous autorise à dire, en son nom, à ce Congrès, qu'il considère la cure de la pneumonie prétuberculeuse par la sérothérapie comme absolue; c'est pour lui un problème définitivement résolu. Or, si le tubercule, dit-il, n'a pas, à proprement parler, de sérum ni de vaccine, la tuberculose spontanée étant forcément précédée de la phlegmasie prétuberculeuse, la vaccination contre cette phlegmasie équivaut à la vaccination contre la tuberculose spontanée.

Cependant nous n'irons pas jusqu'à dire que le sérum peut, à lui seul, guérir toutes les formes de tuberculose. Non, car comme le dit si bien Grasset, le tuberculeux est un malade éminemment personnel, individuel, dont l'idiosyncrasie s'affirme à toutes les périodes et dont le traitement est essentiellement variable.

Nous disons cependant que dans le plus grand nombre des cas et sans autre adjuvant que l'hygiène, la méthode de traitement par les moyens immunisateurs et anti-toxiques donne des résultats réels et souvent définitifs.

Nous ajoutons même que les résultats obtenus avec les sérums et les toxines anti-tuberculeuses, sont merveilleux si on les compare aux moyens curatifs ordinairement employés jusqu'à ce jour. (Baradat, 13ème Congrès International, 1900).

On a eu tort, suivant nous, en niant la similitude de la tuberculose humaine et de la tuberculose animale, de proclamer que les succès sérothérapiques obtenus, dans les laboratoires, sur les animaux tuberculeux, ne pouvaient pas s'accorder avec les conditions spéciales de la tuberculose humaine.

N'avons-nous pas cependant la preuve constante de cette unité, de cette identité dans la découverte même de la contagion de la tuberculose lorsque Villemin fait contracter la tuberculose aux animaux en leur faisant respirer des poussières de crachats humains bacillifères? De même la transmission

Digitized by Google

inverse de la tuberculose des animaux à l'homme n'est-elle pas aujourd'hui un fait demontré? (Boinet, 'Annales d'hygiène publique et de médecine légale.' "Transmission de la tuberculose de l'homme aux animaux et vice versa").

A l'appui de cette dernière assertion, je signalerai incidemment le fait, encore inédit, de la fréquence de la tuberculose pulmonaire *acquise* chez les individus chargés de soigner les singes et les perroquets. Il existe à Marseille, me faisait remarquer, il y a quelques jours, le Professeur Boinet, une oisellerie dans laquelle une série de garçons, employés aux soins des animaux et vivant dans l'atmosphère chaude et contaminée de singes et de perroquets, ont successivement contracté une tuberculose à marche rapide. J'ajouterai aussi le cas d'une famille, sans antécédents tuberculeux héréditaires, sans autre possibilité de contamination, dont les cinq membres sont devenus tuberculeux après avoir dirigé une ménagerie dans laquelle la contamination par les singes, en particulier, ne paraît pas douteuse.

N'est-il pas logique, après des observations aussi précises, d'espérer pouvoir obtenir chez l'homme des résultats aussi heureux que ceux que l'on obtient au laboratoire sur les animaux? Je crois pouvoir répondre par l'affirmative. Du reste, la série heureuse que j'ai observée cette année avec la sérothérapie naturelle antitoxique, employée avec discernement et cliniquement dosée suivant les sujets, me font penser qu'elle constitue la seule méthode rationelle qui puisse être tentée, pour réaliser la guérison de la tuberculose, surtout au début, alors que nos moyens de diagnostic nous permettent de la déceler presque à sa naissance. (Baradat, 'Zeitschrift f. Tub. u. Heilst.' Berlin, juin 1901.)

Il est vrai de dire que sur nos rives de la Mediterranée nous avons pour nous aider dans le traitement de la tuberculose les auxiliaires les plus précieux et les plus puissants, le soleil, la lumière intense, la mer et l'air pur.

Si cette méthode de traitement par les médications immunisantes et antitoxiques n'est pas encore universellement adoptée dans la pratique, c'est en premier lieu parce que son mode d'emploi habituel est difficile et défectueux.

Nous conseillons, en effet, d'abandonner la voie hypodermique pour recourir à la voie interne, stomacale, dans l'administration des sérums et des toxines anti-tuberculeuses. C'est le seul moyen d'éviter chez certaines catégories de malades, chez les nerveux et les alcooliques surtout, ces explosions soudaines d'activités cellulaires, ces manifestations trop intenses de défense phagocytaire qui se traduisent quelquefois par de l'érythème du prurit, en somme, par des accidents que les malades redoutent malgré l'amélioration considérable qu'ils constatent, dans leur état, après quelques jours de traitement.

C'est aussi la voie interne, stomacale, que choisissent Boinet, Grasset et Bertholon pour administrer les sérums et les toxines anti-tuberculeuses; c'est pour eux, comme pour nous, la méthode de choix.

Toutefois nous faisons remarquer que les doses de sérum doivent être augmentées pour obtenir les mêmes effets, toniques et stimulants, et pour compenser l'action des sucs stomacaux sur les toxines utiles; je donne 10

Digitized by Google

cent. c. d'hématégine, preparée suivant la méthode de Bertin et Picq, par la voie interne, tandis que 2 cent. c. sont généralement suffisants en injections sous-cutanées. D'ailleurs la méthode de traitement de Richet et Héricourt par le suc de Viande crue n'agit pas autrement que par l'introduction par la voie stomacale d'antitoxines tuberculeuses.

Ajoutons encore que pas plus avec la sérothérapie antitoxique qu'avec tout autre méthode de traitement, il ne faut attendre qui l'individu soit emacié, qu'il présente des troubles digestifs et de la cachexie pour le soigner.

" En effet, et nous insistons de nouveau sur ce point, un tuberculeux n'est pas un phtisique. Un phtisique est le sujet chez lequel la bacille de Koch, après avoir opéré sa marche progressive, a amené lentement la destruction suppurative des cellules envahies et, dans cette masse détruite, vous rencontrerez tous les processus provoqués par le staphylo, le strepto et le pneumocoque, rongeant, détruisant, parallèlement au bacille de Koch."

" Dans ce cas, c'est la phtisie, comme son nom l'indique, c'est la consomption, la fièvre hectique qui fait mourir ; et supposez pour un instant, que par un pouvoir magique vous arriviez à détruire tous les bacilles de Koch, vos malades succomberont néanmoins au strepto, au staphylo, au pneumocoque."

Je terminerai cette communication sur le traitement de la tuberculose par les antitoxiques par un exposé rapide des travaux très intéressants, mais encore peu connus, de mon savant collègue Bertholon, de l'Institut Pasteur, et par deux observations que je dois à son obligeance.

La constatation de certaines particularités de culture de bacilles de Koch a amené Bertholon à préparer une toxine anti-tuberculeuse qui jouit de la proprieté d'arrêter le développement du bacille de Koch.

En effet, si dans un matras on fait une culture de bacilles de Koch, celle-ci se développe régulièrement jusqu'au moment où on l'additionne de toxine. Elle continue dans les ballons témoins. Après quelques jours, si on essaie de réensemencer les cultures, arrêtées par la toxine, dans un autre milieu de culture, celles-ci ne repartent plus. Elles sont mortes.

Les injections de culture de tuberculose sur des lapins sont arrêtées dans leurs effets par l'emploi de la toxine.

Voici une expérience : trois lapins sont rendus tuberculeux, l'un d'eux meurt. Des deux survivants, on prend le plus malade. Des injections sous-cutanées de toxine sont faites ; le lapin survit, reprend de l'embonpoint. Le mieux portant des deux, *non traité*, meurt.

Bertholon a poursuivi sur des humains une série d'expériences. Ses recherches l'ont conduit à ne plus administrer la toxine que *par la bouche*. Les injections sous-cutanées peuvent amener des accidents ; de plus, la toxine administrée par la bouche détermine chez tous les malades une faim vraiment remarquable. Cette dernière condition est trop précieuse pour ne pas en profiter. Les résultats du traitement sont une diminution rapide de l'expectoration, diminution du nombre des bacilles contenus dans les crachats, disparition des sueurs, signes stéthoscopiques d'induration du pourtour des cavernes.

Parmi de nombreuses observations très remarquables, qu'il nous paraît

Digitized by Google

superflu d'énumérer ici, nous en résumerons deux qui nous paraissent d'autant plus intéressantes qu'elles se rapportent à des formes de tuberculose très avancées sur lesquelles l'action thérapeutique n'a jamais eu de prise jusqu'à ce jour.

Obs. I.—B., *ouvrier*, vaste caverne au sommet droit, cachexie, suffocations, diarrhée, expectoration abondante avec bacilles nombreux, *oedème des extrémités inférieures.*

Le traitement par la toxine anti-tuberculeuse l'a suffisamment remonté pour qu'au bout de trois mois et demi il ait pu reprendre son travail, tout en toussant encore. Il continue son traitement. Le pourtour de sa caverne présente des symptômes d'induration. Si cet homme était riche et *pouvait se reposer* il guérirait vite et sûrement.

Obs. II.—Voici un autre cas non moins curieux; il s'agit d'une fillette de onze ans. Cette enfant, tuberculeuse depuis un an, avait fait de la pleurésie purulente avec pus contenant le bacille de Koch. On lui avait pratiqué un Estlander à l'hôpital militaire de Tunis. Les médecins militaires, la voyant parvenir au degré ultime de sa maladie et ayant abandonné tout espoir, avaient conseillé au père, en février 1901, de la reprendre chez lui pour qu'elle mourût au milieu de sa famille. Elle avait alors une diarrhée avec 25 selles par jour, une expectoration de tous moments; c'était un squelette.

Cette enfant est soumise au traitement par la toxine depuis 6 mois; sa diarrhée tuberculeuse a guéri dès le début; l'expectoration est supprimée; elle a perdu son état squeletique; elle se nourrit bien et reprend des forces. Le seul point noir est l'insuffisance de l'opération d'Estlander qui entretient une fistule pleurale.

Bertholon débute dans le traitement par sa toxine anti-tuberculeuse, par une dose de 1 c.c. de solution au 1/20 de toxine dans la glycérine.

Le remède est administré tous les trois jours, puis plus fréquemment. L'observation journalière des malades et la manière dont se comportent l'appétit, l'expectoration et enfin l'état local des poumons sont des guides qui permettent au médecin de varier les doses.

Dans tous les cas, c'est un moyen thérapeutique dont il faut surveiller de très près l'administration.

Les faits que nous apportons nous permettent de conclure que, dans des cas determinés, lorsque la tuberculose est à son début, la méthode par les sérums ou par les toxines anti-tuberculeux, ceux-ci étant introduits par la voie buccale, est appelée à rendre de précieux services; les expériences poursuivies parallèlement chez l'animal et chez l'homme, susceptibles de se transmettre réciproquement la tuberculose, ont donné des résultats identiques et permettent actuellement de fonder la méthode, que nous préconisons, sur des bases sérieuses.

Digitized by Google

THE INHALATION OF FORMIC ALDEHYDE AS AN AID IN THE OPEN-AIR TREATMENT OF PULMONARY TUBERCULOSIS.

By CHOWRY MUTHU, M.D. Brux.,

Resident Physician, Inglewood Sanatorium, St. Lawrence, I.W.

Formic Aldehyde.—The discovery of micro-organisms as the exciting cause of disease has brought into prominence many antiseptics and disinfectants, the object of which is to check their growth or destroy them. One of the latest of these is formic aldehyde, which is obtained by the oxidation of methyl alcohol.

A great amount of experimental work has been carried on on the Continent, in America, and in England, which goes to prove that formic aldehyde is a powerful antiseptic and germicide, its bactericidal action being equal to, if not greater than, that of bichloride of mercury.

It is at present very popular among the medical profession, its use being extended every day in the fields of histology, surgery, medicine, and hygiene.

It is rapidly superseding the older antiseptics, such as carbolic acid, perchloride of mercury, and others. Its advantages are :—

1. It is non-poisonous and non-corrosive.
2. It can be used in the form of vapour, which, being of low specific gravity, is diffusible, and mixes well with the air.
3. It has no action on clothes, textile fabrics, leather goods, and general household articles, or on metals, except iron and steel.
4. It is easy and clean in its application.

F. Aldehyde in the Disinfection of Rooms.—Besides being used as a general antiseptic and deodorant, it has been largely employed in the disinfection of rooms with success.

About two years ago, in the course of some experiments, I made the following observations :—

1. Linen strips dipped in broth cultures of staphylococcus pyogenes aureus, and exposed in a sealed room with a capacity of 2000 cubic feet to the vapour generated from twenty paraform tablets, showed no growth after six hours.
2. Half to one per cent. formalin solution killed a pure culture of staphylococcus pyogenes aureus bacillus anthracis, bacterium coli communis, bacillus typhosus, within thirty minutes to an hour; and ever since I found formaldehyde to be most efficient and reliable in the disinfection of rooms in the sanatorium with which I am connected.

Digitized by Google

F. Aldehyde in Pulmonary Tuberculosis.—The next step in the progressive use of formaldehyde has been its application in the case of throat and lung disease—especially in pulmonary tuberculosis—in which disease encouraging results have been obtained by medical men.

Mr. Lardner Green, of Salisbury, was almost the first in this country to bring before the medical profession the desirability of using the inhalation of formaldehyde in early cases of pulmonary tuberculosis.

Dr. William Murrell, of London, in his report to the Scientific Grants Committee, 1899, related twelve successful cases that have been treated with the vapour.

On the Continent, Professor Cervello, of Palermo University, has given considerable attention to this subject, and has invented an apparatus for the production of formaldehyde gas.

My Observations.—I began treating patients with the drug in 1899, and have continued my clinical observations ever since.

I have already published the results of *seven cases*, which are briefly as follows: Three were cured, two were benefited, and in two others the results were not conclusive.

I have now to record the results of *fifteen cases*. Before so doing a few words as to the apparatus used would be desirable.

Formaldehyde was used :—

1. In the form of gas, and
2. Formalin (40 per cent. solution).

The gas was generated in two ways :—

(*a*) *The Dry Method.*—In this method paraform tabloids—called dry formalin—were placed on a metal tray over a methylated spirit lamp (alformant lamp).

The principle of this method is that the hot moist products from the combustion of the spirit, acting upon the tabloids, convert them into formaldehyde gas.

(*b*) *The Moist Method.* In this method, in addition to the above apparatus, there is a boiler for generating steam, which, mixing with the formaldehyde vapour, renders it more diffusible and penetrating in its action. Of these two methods the latter is the more efficacious.

Method of Using.

1. The vapour is administered either in the patient's bedroom or in the inhalation room, which faces south, having plenty of sunshine; for the drier the atmosphere, the more the vapour is generated, the greater its penetrating power. The doors and windows are partially or wholly closed, the lamp is lit, the boiler is filled with warm water, the paraform tabloids are put in, the patient sits in a lounge chair or lies in his bed and inhales the gas.

Digitized by Google

The vapour at first causes irritation of the eyes and nostrils, which passes off after a few minutes. Th inhalation is continued for one, two, or three hours, when the doors and windows are again opened.

2. The aqueous solution of the vapour or formalin (40 per cent.), as it is called, is used in two ways.

(*a*) It is used (6 to 10 per cent. solution) in an inhaler, which the patient places over his mouth, using it from four to six hours in the course of the day.

(*b*) It is also used in the form of fine spray, or in a nebuliser, at a strength of 6 to 10 per cent. solution mixed with glycerine.

The patient uses both these methods in the course of the day, either when he is in the open air or in his day shelter with doors and windows wide open.

Results.

Fifteen patients (twelve men and three women) have been treated in this way for three to five months. All of them have been my patients in the Sanatorium, undergoing the open-air treatment for six to eleven months. They have not had any special medicine beyond an occasional pill or a mixture to correct indigestion, consequently the results have not been complicated in any way by the treatment.

Of the fifteen patients:—

Five (all men) were completely cured. No physical signs or bacilli were found when they left. They have ever since continued to be in good health.

Seven (six men and one woman) were almost cured; *i.e.*, a few dry crepitations and a few bacilli remained when they left, but they have been in good health and have followed their different avocations.

One (a woman) was slightly benefited.

Two, in the rest it had very little effect.

Of the five who were completely restored to health, three had affection of one lung, one had a large cavity in the left apex, the other had marked signs of breaking down of both lungs.

Of the seven who were nearly cured, two had cavities of one lung and consolidation of the other, one had empyema complicating pulmonary tuberculosis, and the rest had more or less extensive affection of both lungs.

Digitized by Google

Some Typical Cases in Detail.

Remarks.

1. The results of the treatment, to my mind, have been very satisfactory, and I would strongly urge that the drug should be given a fair trial in every sanatorium where the open-air treatment is carried out.

2. It should be systematically and persistently used; and some one enthusiastic in the treatment should take charge of the apparatus, for the apparatus requires to be kept clean, and the lamp, etc., need careful supervision every day; any neglect would bring about indifferent vaporisation, and the results will be more or less a failure.

3. I do not claim that formalin is a specific, or that it is beneficial in every case. What I do believe is that, if judiciously and persistently administered, the inhalation of formaldehyde will prove to be an important aid in the cure by the open-air treatment. There are two important principles we keep in view in the treatment of pulmonary tuberculosis. The first and more important is to so strengthen the system as to increase the resisting power of the tissues and cells of the body, which are our real friends in combating the enemy. This is done by fresh air, careful feeding, judicious rest, regulated exercise—four cardinal points in the open-air treatment. The second is to introduce an antiseptic directly or indirectly into the system which in some way will inhibit the action of the bacilli or their toxins. I believe that formaldehyde belongs to the latter category. When the two methods are used together the cure of the patient will be rendered more certain and his stay in a sanatorium shortened. Its beneficial effect is specially seen in cases of cavities and in empyema complicating pulmonary tuberculosis. How it acts in the body, whether its antiseptic properties inhibit the growth of the bacilli or neutralise the action of the toxins, or whether it merely prevents secondary infection, is still uncertain. Dr. Maguire, of Brompton Hospital, has reported favourable results from the injection of formaldehyde (1 in 2000) directly into the vein. This perhaps is not without a certain amount of risk at present. Further proof will be necessary before this method of intra-venous injection becomes of general use. But whether by inhalation or by some other and more heroic method which future research will make clear, I believe that in formaldehyde we shall find a very important remedy in the cure of pulmonary tuberculosis.

Digitized by Google

TREATMENT OF PULMONARY TUBERCULOSIS BY MEANS OF FORMALIN INTRAVENOUS INJECTIONS.

By R. MAGUIRE, M.D. Lond., F.R.C.P.,
Physician to the Brompton Hospital, London.

At the end of last year I published in the Journals a preliminary announcement of the results I had obtained and of the experiments I had made on a method of treating pulmonary tuberculosis by rational means. It will save time, and help us to understand the additional records I have to give you, if I now very shortly review the arguments I then advanced in favour of the method I have adopted.

We wish sincerely for an antiseptic fluid, similar to that which we now use for diphtheria, which, being injected into the tissues in small quantity, will neutralise the effect of the tubercle bacillus, kill it *in situ*, and destroy its effects. Some day we shall obtain this, but up to now Professor Koch's tuberculin has not fulfilled our expectations, and we must try other means to cure our patients.

The anatomy of the lungs is peculiar. Everything which obtains access to the veins must necessarily be carried to the lungs; this is true of all the veins of the body but those of the portal circulation. These carry the blood, first to the liver and then to the lungs, altered, meanwhile, by the action of the liver. But the peripheric veins carry everything to the lungs. Upon this fact I have worked. By this method and way I have trusted to apply medicaments to the lungs and to treat those organs exactly as the surgeons treat external organs—that is, locally and directly.

Tuberculosis of the lungs is due, primarily, to infection by the tubercle bacillus, but assisted also by minor germs, the staphylococcus pyogenes and the pneumococcus. Our object should be to poison the tubercle bacillus, and if we cannot do so much, then to kill its congeners and let Nature then finish the work. At the present time most efforts are directed to the increase of the resistant power, and rightly so; but what about the cases which will still occur, not only for a few years, but long after our time, and which will demand treatment more than the open-air sanatoria can yet give? In the midst of our researches after a panacea, we must not ignore the actual fact that we have many cases of tuberculosis of the lungs who appeal to their doctors for relief. Let us descend from the clouds and be practical.

In order to kill the tubercle bacillus, or any other germ in the lungs, it is useless to try inhalations. Nothing strong enough to kill a germ could possibly pass the glottis, and still less, even if it could proceed farther than the large bronchi, could it penetrate the cells in sufficient strength to be germicidal.

Given by the mouth it must still more be diluted in the stomach and altered in its passage through the portal circulation.

Digitized by Google

Administered through the skin it must be diluted by all the fluids of the body. And, if we wished to apply it directly by perforation of the chest to localised spots of tuberculous infection, it could never have any great degree of diffusion, and no one could say what other areas of tuberculous pneumonia would escape the operator's needle.

Why then not sluice out the lung by a germicidal fluid and by means of the peripheric veins, if only this can be done with safety?

I have tried to do this, and, as I hope to show you, I have done so with safety and success.

Applying medicaments to the lungs locally, and by the means of intravenous injection, I tried first to use a solvent to the cellulose envelope of the tubercle bacillus in the form of diastase, and afterwards of cytase. These are efficient in the laboratory, but of no use in clinical practice. I tried nuclein, with the view of stimulating the phagocytes. This, by the mouth, has a marvellous action, as I found on tuberculous glands, but by the mouth, by the skin, and even intravenously, it had no good effect upon tuberculosis of the lungs. Further, all these agents produced a decided febrile reaction, resembling a malarial attack, which could not but be harmful to a weak tuberculous patient.

But in trying, and with great difficulty, to make the solutions of these substances aseptic, and fit for intravenous injection, I found that a pure solution of formic aldehyde was the best medium, and at last came to the conclusion that this, used intravenously as an antiseptic, was safe and likely to be efficacious when used alone. I need not here enter into the details of experiments I made on animals and myself to determine the exact strength to be used. Suffice it to say that my colleague, Mr. Plimmer, tried on rabbits, and I tried on myself, the effect of each individual strength of solution before subjecting a patient to its effects. In the earlier stages certain unpleasant symptoms occurred in me, which have been greatly magnified and misunderstood. Since then, and by improved methods, I have injected as high a strength as 1 in 500 into the human being and 1 in 200 into a rabbit without ill-effect, provided that, for the human subject, the quantity of 50 c.c. be not exceeded. For general use a strength of 1 in 2000 is best, and of this 50 c.c. should be injected daily by means of the apparatus shown on the table In doing this with all proper precautions there is not the slightest dange. whatever, and upon this I insist, as many rumours to the contrary have got about.

But the technique is by no means easy to acquire. It demands practice, deftness of hand, and an acute sense of touch—naturally, too, a knowledge of anatomy and caution. Time will not allow me to enter into all the little hints which might be given. I tie up the arm with a silk handkerchief, get the patient to squeeze hard upon a folded towel—the "barber's pole"—lave the inner part of the elbow with 1 in 20 of carbolic acid, and anoint it and the needle with 1 in 20 of carbolic vaseline. Then fixing the elbow as firmly as possible, I pierce the vein with the needle almost vertically, dropping the wrist when once the inside of the vein has been reached. The needle then

Digitized by Google

plunges into the vein suddenly—a sensation which is indescribable—and the operator must be extremely careful not to perforate the hinder or hither wall. The "barber's pole" being relaxed and the bandage untied the current of fluid may be allowed to flow. If the operation is successful, a little gurgle is heard as the fluid tumbles over itself—and all goes well. The pain at the site of injection is not to be thought twice about; but sometimes, from, as I think, an irritation of the nervi vasorum, there is occasionally a little aching up one or other side of the arm and a cramp-like sensation in the fingers, all passing off in about three minutes. Thus, if matters are successful. But if the operator be inexpert or unfortunate he may pierce or miss the vein. Then there is a swelling at the site of injection, considerable temporary pain, and perhaps some subsequent inflammation. Watchfulness will detect the mishap and stop the extravenous effusion as soon as possible. Another vein should then be tried, preferably in the opposite arm, and in any case opposite arms should be used on alternate days. If all these precautions be attended to, and also the strength of solution I have mentioned used, I repeat that there is not the slightest danger in the process.

Once only have I had a serious mishap, and that was when, as I now think, ill-advisedly, I had added absolute alcohol to the injection fluid, and in a very difficult case I produced neuritis.

As to the solution to be used, I soon found that in general composition it must be made to correspond as nearly as possible with the serum of the blood, and after many experiments came to the conclusion that its best basis was a 7 per 1000 solution of NaCl, to which pure formic aldehyde was added, so as to make a strength of 1 in 2000. For special cases I have used a strength of 1 in 1000, and even 1 in 500. In no case should a greater amount than 50 c.c. be injected at one time. Used in this way no danger whatever can ensue. The solution must be made absolutely sterile. This might seem needless, as the formic aldehyde is itself an antiseptic. But it must be remembered that in this strength formic aldehyde is not noxious to the mould fungus, and precautions must be taken to prevent the access of this to the veins. As nearly as possible the solution should be kept at blood heat, warmed in winter (for a cold injection is painful), cooled in summer, to prevent the oxidation of the aldehyde to alcohol. I may briefly state that the formic aldehyde passes through the lungs without change of any great extent, for I have found it in the urine, the sputum, and the exhaled air after injection.

We can also calculate in what strength the injected fluid reaches the lungs by counting the number of heart beats which occur during the period of injection. Using a large needle, and in a quiet non-excitable subject, I have succeeded in injecting 2 c.c. of a 1 in 1000 solution for each heart beat. Taking the right ventricle to contain 100 c.c. we should then have a solution of formic aldehyde in that cavity of 1 in 50,000—a very potent germicide, and indeed stronger than is required. In order to maintain as great a strength as possible in the lung blood-vessels, the pulse should be kept quiet, and the patient should be in the recumbent position.

Digitized by Google

We ought now to inquire what we should look to to show the effects of the injections upon the tuberculous process in the lungs, and they are, I think, the following: Abolition of pyrexia, extinction of physical signs, increase of appetite and general well-being, and an alleviation of the sputum, ending in—the final test of cure—the disappearance of the tubercle bacilli. I can with confidence say that in favourable cases, and in not a few of unfavourable cases, all these results have been and can be obtained. Before entering into details I may state here that of the last twenty cases I have treated privately, some of them even in an advanced stage, only two have failed to show the disappearance of the tubercle bacillus, and both these cases were desperate and apparently hopeless, and in one I was obliged to cease the treatment because of the extreme difficulty in getting entrance into the veins. It must be remembered, too, that these cases were treated in London and in lodgings, with scarcely any attempt at open-air treatment more than a simple opening of the windows. Not that I would in any way decry the open-air treatment properly pursued in sanatoria, but apart from the larger numbers of patients whom I have treated, the results of these twenty consecutive cases, details of which I will presently give, show most favourably when compared with those of the sanatoria. To get statistics from the English sanatoria, most of which are private ventures, is very difficult; but Professor Koch stated here a few days ago that in the German institutions 20 per cent. only showed a disappearance of tubercle bacilli from the sputum, and it must be remembered that if the whole of the nearly 200 cases I have treated myself had been under better surroundings the results would have been much better.

In estimating the value of any given treatment, it is most important not to deceive oneself. One would naturally choose early cases in which to perform a cure, and I will presently show you results of these. But at the beginning of my observations I wilfully chose desperate cases, for which otherwise no relief could be given, and of these even I shall have to show you records of the checking of the tuberculous process. A few such cases are more demonstrative than many relieved in the earlier stages, but it would be manifestly unfair to compile statistics which included such hopeless patients.

I have, as I have stated, now treated nearly two hundred cases, some privately, some in hospital, some with the assistance of Dr. Reid as out-patients in the poorer parts of Lambeth, and while they were at work and living in their very undesirable surroundings, some with Dr. Findlater in the Hendon Infirmary in what is practically the country. Many other medical men have treated cases by my method, and amongst others especially Dr. Hyla Greves, of Bournemouth, so that now the numbers of accumulated results warrant one in drawing conclusions.

I would classify the cases as follows, and show you examples of the records of each:—

I. Cases in an early stage, showing disappearance of all symptoms and signs, and of tubercle bacilli in the sputum.

II. Cases in the state of cavitation, showing disappearance of all symptoms

Digitized by Google

and of tubercle bacilli in the sputum, but with the remnant of physical signs in the form of a dry cavity.

III. Cases in the stage of cavitation and of very active tuberculosis, with much destruction of lung tissue, in which symptoms and signs greatly diminished in amount, but tubercle bacilli did not disappear from the sputum.

IV. Desperate cases which ended in death, but showed previously some relief from the symptoms.

ON TIME-SAVING METHODS OF TREATMENT, WITH A PRELIMINARY NOTE ON THE "SILVER TREATMENT" OF PHTHISIS BY INTRAVENOUS INJECTIONS OF PROTARGOL.

By WM. EWART, M.D. Cantab., F.R.C.P.,

Physician to St. George's Hospital and to the Belgrave Hospital for Children, London.

It is now recognised that phthisis is curable by the open air if sufficient time be allowed. Our next effort should be to shorten the cure, and this communication is an endeavour to promote the search for more expeditious methods, without which the complete realisation of the scheme of State-aided relief cannot be carried out, owing to the vast numbers to be dealt with and to the costliness of the necessary accommodation and of the treatment and diet. Much, therefore, would have been achieved if the individual stay at State sanatoria could be shortened; and this achievement is claimed as possible if cases of phthisis (excluding always hopeless cases) were to be provided with adequate preliminary treatment, such as to fit them to secure from sanatoria the maximum good in the minimum time.

Quite recently strong claims have been put forward in favour of various methods of treatment. Special notice would seem to be due to those with pretensions as "specifics," most of which probably do some appreciable good, even though not fulfilling their entire promise. A strong plea must therefore be entered in the interest of the rate-paying public, as well as of the patients, for an adequate inquiry into the comparative capabilities of the various "cures" now available. It is clear that we cannot rest satisfied with our present system, however great its advance upon previous conditions.

The "Thorough Treatment" or "Hospital Treatment" as a preliminary to Open-Air or Sanatorium Treatment.—Early and mild cases in strong subjects are practically self-curable with an open-air life; but with the large group of pyrexial, sub-pyrexial, or more or less chronic phthisis, the open-air treatment, at any rate in this country, and also the climatic treatment, so it would seem from Alpine experience, is not the shortest way to a cure. Nay, many cases are at first ill-suited for it. In these cases progressive tuberculosis is perpetuated by the catarrh. A great deal of the latter is remediable by what may be termed the hospital treatment, or "thorough" treatment, the object of

Digitized by Google

which is to rid the case as soon as possible of its dangerous complications—bronchitis, catarrh, caseation, and suppuration—whilst utilising the period of unavoidable confinement in bed, in the ward, or in an even temperature, for the application of every therapeutical influence which in the individual case may be profitably resorted to. This idea has been recently carried out with an encouraging degree of success in a limited number of cases. The favourable results obtained have demonstrated to my satisfaction the advantage of not sending the patients to the open air prematurely, but only in a convalescent state.

The Direct Pulmonary Methods, including the Intravenous.—In addition to serum-therapy, which belongs to the future, these include the *intra-tracheal injections*, which are not sufficiently recommended by their results; the *inhalation treatment*, well known to all in its more familiar applications, but specially elaborated into definite methods by Cervello with only partial success as the formaldehyde system, and more recently with every promise of practical usefulness by Dr. George Stoker as the *continuous nasal oxygen inhalation method;* and, lastly, the *intravenous injections*, which in some form, perhaps quite different from any at present in use, are likely to prove the most active and rapid agency. *Landerer's sodium cinnamate treatment* has had a fair trial, and the bulky injection method which we owe to Dr. Maguire's labours is now being tried. I am not acquainted with the latest development of *the formaldehyde injection treatment*, but some of the results obtained with the original method were very favourable, although neither by this nor by any other method has a permanent clearance of the bacilli from the tissues been obtained.

I was led to substitute *protargol* for formaldehyde by the conspicuous success of the administration of nitrate of silver in pneumonia (Caccianiga) and of its subcutaneous injection in phthisis (Mays). Pending the discovery of some better remedy I have provisionally adhered to it in preference to formaldehyde because of its more decided, of its more rapid, and of its more lasting action. The clinical effects resemble those obtained by Dr. Maguire with formaldehyde—*viz.*, a remarkable subjective feeling of improvement coinciding with manifest improvement in aspect and in strength, a rapid diminution in the cough and in the expectoration, and a more or less rapid change from the dense sputum of phthisis to simple catarrhal sputum and ultimately to hyaline mucus, etc.

The ultimate effect is to lower the temperature, but meanwhile the level may be disturbed by elevations incidental to the treatment. More often than not the injection is followed after one hour by a chilliness or even a rigor and a sharp rise lasting about half an hour. After this has passed off the patient feels and continues to feel remarkably well; no untoward symptoms of any kind have followed the rigors. A short attack of nephritis occurred in one patient, but this seems to have been occasioned by a chill. Pre-existing albuminuria of long standing was much reduced in one of my cases; in another the first injection succeeded in stopping a rebellious hæmoptysis.

Digitized by Google

The Technique.—The details of the operation are given in the paper. The injection consists of 40 cubic centimetres of saline solution containing 1½ to 2½ grains protargol, and this is preceded and followed by an injection (through the same needle) of a few cubic centimetres of pure saline solution to obviate leakage of irritating fluid into the tissues, which is apt to lead to considerable pain and swelling. From twelve to fifteen injections generally suffice. It is best to administer them at intervals of one day, but in some cases they have been given daily.

The Systematic Treatment.—The protargol injection method has yielded by itself satisfactory results, but it does not claim to be more than the first and most important instalment in an extensive system of active treatment. My present practice is to combine with it, as soon as the case has lost all acuteness, general massage, gentle exercise, and particularly respiratory exercise for the expansion of the lung, strong diet and nutrient adjuncts, local and general treatment of the skin, and suitable internal remedies, among which I have obtained specially good results from ichthyol, originally recommended by Wertheimer. This combined treatment, a full description of which cannot be briefly given, has enabled some patients with originally unfavourable prognosis to become, after a very few weeks' stay in the hospital, quite suitable for the open-air treatment, and to return, after a short period in the country, greatly improved, and with a promising forecast of ultimate recovery.

Drugs may have been too much discouraged owing to the superior virtues of "open air." Our knowledge of the latter should not paralyse, but stimulate our therapeutical activity. A place is still to be found for the old remedies, not any longer as the sum of our treatment, but as useful or necessary adjuncts. Expectorants, antipyretics, tonics, and sedatives may all have their temporary uses. But there are new remedies from which greater help may be derived, particularly when judiciously combined. Good combinations have been sacrificed to the desire to prove any one system to be a specific cure. The *continuous inhalation of oxygen*, for instance, is compatible with various hygienic helps, and with most useful forms of medication. The same is true of the *hyper-nitrogenous alimentation* advocated by Richet (raw meat treatment) and by Harper (urea treatment).

Wertheimer's ichthyol treatment consists in the administration after meals of a few drops of ichthyol, say in peppermint water, with a daily increase of one minim till ten minims are taken—some of my patients have taken twenty minims with advantage. A diminution in the cough and expectoration, and a change in the character of the sputum, are perceptible in a few days. My own experience endorses Wertheimer's most encouraging reports, and for the present I have no hesitation in recommending ichthyol as the remedy which will most favourably and most rapidly influence the catarrhal complication, even when administered singly. But this or any other medicine found to be superior may be used in combination with intravenous injections; and it is my belief that such a combination would help us to effect a rapid change in

Digitized by Google

the condition of the lung, and to subdue the catarrh sometimes within two to four weeks, and thus pave the way for a rapid improvement under the "open-air" treatment.

Conclusions.—In this system of treatment there is nothing final. On the contrary, all its details are provisional and merely the best that existing opportunities afford. Its results, hitherto, seem to justify a hope that yet better things are in store, and that well-sustained efforts in the direction indicated will, at no distant date, bring about a considerable reduction in the total duration of the cure of phthisis, and in the minimum period to be spent at sanatoria.

The cases treated have all been hospital cases, and therefore illustrate the possibilities open to the State for the treatment of phthisis among the poor on a considerable scale, if poor-law infirmaries and sanatoria were thoroughly organised for carrying out a well-conceived systematic treatment. In all its details, and in special connection with the intravenous injections, the general scheme which has been proposed could be more easily conducted at public institutions than in private.

So long as no better remedies are forthcoming, those which have been suggested may be used with safety and profit; but it is urgent, in view of the important issues at stake, that a systematic inquiry should be undertaken into the comparative value of the various forms of treatment elaborated by individual efforts, and for some of which important claims have been advanced.

THE TREATMENT OF TUBERCULOSIS BY UREA.

By ARTHUR H. BUCK, F.R.C.S.,
Assistant Surgeon, Sussex County Hospital.

It is, I take it, generally granted that the most important factors in dealing with any cure for tuberculosis are susceptibility and immunity.

Jenner first recognised the importance of the immunity of a portion of the community to a particular disease, and eventually his labours have procured the immunity of the rest from that disease.

It was not, however, till comparatively recently that the importance of his work was understood.

All the great workers in this direction, whilst studying susceptibility and immunity, have tended to be drawn in one direction, and that is the preparation of antitoxins, by means of the injection of which into the human body we can either render such immune to, or modify an attack already begun, by the germ peculiar to each disease.

These antitoxins have been prepared from the growth of the different organisms concerned, under various conditions, and the result is that the chemical substances which have been injected into the tissues of those to be protected are powerful alkaloids or toxalbumins differing chemically from any combinations hitherto demonstrated in our organisations.

Digitized by Google

Now this class of antitoxins have so far proved of greater value in acute specific fevers, such as diphtheria, small-pox, typhoid and bubonic plague, than has the corresponding antitoxin in tuberculosis. But tuberculosis differs in many other respects from acute specific fevers. The former is a chronic disease, the latter acute.

The tubercle bacillus is one of the family of organisms which are termed infective granulomata; the latter belong to a class which produce their toxic effects in the body without the formation of new growth around them.

The tubercle bacillus has an exceedingly wide distribution; that of the others is, comparatively speaking, extremely limited.

Every one of us has been exposed, some constantly, to infection by tubercle, comparatively few have developed the disease; whereas of those who, being unprotected either by a special antitoxin or by a previous attack, are exposed to active infection by any of the acute specific fevers, I presume it is safe to say that the great majority would develop such.

In other words, by far the greater proportion of mankind provide a fit soil for the growth of the germs of acute specific fevers, whereas a small minority only are capable of harbouring and developing in their tissues the tubercle bacillus.

The majority of us, therefore, possess in our systems some vito-chemical combination or condition which renders us immune to any ordinary dose of infection by the tubercle bacillus, and it follows that that combination or condition must be actively hostile to the bacillus, for we are all exposed to the infection.

This substance or condition, therefore, whatever it may be, in contra-distinction to the antitoxins, must be such as is neither harmful nor foreign to the human organism. It most probably exists in some in a greater degree than in others; but in any case, if we could induce in the tissues of the susceptible some difference in metabolism, so that this substance or condition was introduced into or manufactured in their systems, there can be no manner of doubt but that we should rid the human race of tuberculosis.

It was by the observation that in his experience the gouty were particularly immune to tubercle that induced Dr. Harper, of Nottingham, to prescribe pure urea to the tuberculous with a view to increasing the amount of urea and uric acid in their systems, and it is because of the enormous importance that I believe attaches to this treatment that I am reading this paper.

Dr. Harper has published two papers full of interest in the *Lancet* this year (March 9th and June 15th), in which he mentions many cases of phthisis which, being apparently incurable, have recovered. I would draw attention to the fact that Dr. Harper was induced to try this treatment by the observation of the peculiar immunity of a small part of the community, namely, the gouty, to tuberculosis; in other words, for reasons similar to those which induced Jenner to try vaccination for small-pox.

I think it is an extremely interesting fact to note that urea, in the shape of urine, has been taken from time immemorable as a medicine, and that the natives of India are at present using it in this way.

On the books of the Calcutta Zoological Gardens is an item, in the

Digitized by Google

receipts, of £60 annually paid for the urine of the rhinoceros, which, I am informed by the gentleman who told me of this fact, the natives buy and take internally in cases of lung disease.

Does urea benefit cases of tuberculosis; if so, how and why?

It is extremely hard to convince anyone of the value of a drug who has not tried it himself, but from the extraordinary results I have myself seen I have not the slightest doubt in answering the first question with a decided affirmative, as regards some cases.

As the treatment is still on trial and in its infancy, the answer to the second must necessarily be uncertain. The following facts, however, will, I believe, be found to have a bearing on the case.

Urea is one of the end products of nitrogenous metabolism, *i.e.*, of the oxidisation or reduction of proteid matter, and the medium in which most of the nitrogen excreted from the body is carried away is the urine. Its chemical formula is $CO(NH_2)_2$, and it is isomeric with cyanate of ammonium $(NH_4)CNO$, from which it can be prepared synthetically.

Urea is formed, according to our latest knowledge, in the spleen, lymph and secreting glands, but principally by the liver. It is excreted by the kidneys.

Urea has lately been produced from proteid material in the chemical laboratory, but by processes which cannot be the same as those which go on in the body.

The original source of the nitrogenous matter which eventually supplies urea is, of course, the proteids consumed as foods.

"These proteids are the most important substances," to quote Dr. Halliburton, "that occur in animal or vegetable organisms. None of the phenomena of life occur without their presence;" and yet we do not know their exact constitutional formula, nor, indeed, do we know whether they exist as such in living protoplasm, "though they are always obtained by subjecting living structures to analytical processes."

"All that we know with certainty is that many different substances may be obtained by the decomposition of proteids. How they are built up into the proteid molecule is unknown. The decompositions that occur in the body are, moreover, different from those which can be made to occur in the chemical laboratory, hence the conclusion that living protoplasm differs somewhat from the non-living proteid matter obtainable from it."

With this state of uncertainty it is evidently impossible at present to understand precisely why and how urea can act beneficially in tuberculosis, or what changes exactly take place on its re-entrance into the body. Again we can only deal with facts.

Urea contains nitrogen, as do these all-important proteid matters.

During the period of growth and development less nitrogen is eliminated from the body than during the decline of life. The balance of nitrogen, therefore, with absorption at one end of the scale and elimination at the other, plays an important part in the cell activity and vito-chemical constitution of the body. If we can prove that urea splits up during the

Digitized by Google

process of absorption, and that its nitrogen is assimilated, it can only be assimilated in the metabolism of protoplasm. We shall then have established the curious fact that an apparently waste product, the result of a process of reduction by oxidisation of protoplasm, on being passed through the mill, so to speak, a second time, assists in building up tissues similar to those from which it was originally derived. The proof that urea is decomposed in the process of absorption and assimilation, and that its nitrogen is wholly or in part retained in the body, can only be ascertained with certainty by elaborate processes of analysis.

With the general practitioner this is, as a rule, impossible, owing to lack of experience, time, and apparatus. But more experiments, including estimation of total nitrogen eliminated, will be necessary before definite statements can be made. Dr. Harper mentions in his paper that the urea in most cases is not increased. I append a few notes on this subject which I have obtained, from which it will be seen that the urea is more often below normal than above. For the estimation of the uric acid I have to express my best thanks to Mr. Gomess, of South Kensington, and for that of the urea to Mr. Tweedy, of the Sussex County Hospital.

Dose Urea.	Average Total Urine.	Average Urea.	Uric Acid.	Age of Patient.
1. 30 grs. t.d.s . .	43 oz.	2·1 per cent	·04 per cent.	32
2. 30 ,, ,, . .	50 ,,	1·9 ,, before taking urea 1·6 ,, after ,, ,,		55
3. 1 dr. ,, . .	43 ,,	1·5 ,,	. . . .	36
4. 20 grs. ,, . .	33 ,,	2·5 ,,	. . . .	12
5. 40 ,, ,, . .	Uncertain.	·7 ,,	. . . .	22
6. 30 ,, ,, . .	,,	2·2 ,,	. . . .	18
7. 20 ,, ,, . .	35 oz.	2·5 ,,	. . . .	12
8. 25 ,, ,, . .	36 ,,	1·4 ,,	. . . .	15

Mr. Gomess has also very kindly worked out the total amount of nitrogen in the first two cases, with the following result:—

1st. By Kjeldahl's method 5 c.c urine = 37·8 c.c. nitrogen.

2nd. By Kjeldahl's method 5 c.c urine = 36·4 c.c. nitrogen.

Total amount of nitrogen in 45 oz. urine in first case equals 8640 c.c., and in 50 oz. urine in second equals 9245 c.c. Mr. Gomess adds, "My own opinion is, if I may judge from above figures, that the nitrogen eliminated in the urine is much less than the amount given in the form of urea."

The percentage of nitrogen in urea to that in flesh is as 46⅔ to 16. It will be seen that the nitrogen taken in the food has to be added to that taken in the urea, and it seems plain that the extra nitrogen has not been excreted in the urine, at all events in the two cases quoted above.

A short description of seven cases which I have treated in this way is as follows:—

Case 1.

Man, aged thirty-six. Was deserted as an infant, and suffered from tuberculous disease of left ankle and both elbow joints during childhood.

Digitized by Google

Amputation was performed below the knee for the ankle joint disease, and double arthrectomy of elbows at age of ten to eleven.

Twelve years ago lupus commenced in nostrils and spread with some rapidity to face and forehead. In spite of various treatment the disease progressed, and the following was condition when on 26th March last I commenced treatment with urea.

One large ulcer occupied an area, around which I will trace an imaginary line commencing at the centre of hair of forehead to a point half an inch above the outer end of each eyebrow, then inwards along the upper border of the eyebrows to a point above the root of the nose. The nasal bones had necrosed and come away, leaving a round hole leading into nasal cavity. Ulceration existed on both cheeks, and the left lower eyelid had gone. From the outer edge of the ulcer on one cheek to a corresponding point on the other measured six and a half inches. In the centre of the face was a bridge of tissue, representing remains of the nose covered with epithelium. Below this was another irregular hole where had been the nostrils. Ulceration was present over the area where the upper lip had existed, most of this having disappeared. The edges of these large ulcers were very much raised, irregular nodules composed of chestnut-coloured, apple-jelly-like matter, sharp cut towards the ulcer and gradually sloping down towards healthy skin. Some of the nodules stood three-sixteenths to a quarter of an inch above the level of the ulcer.

Inside the nose one large cavity existed, the septum, part of the ethmoid and most of the turbinated bones having disappeared. The walls of this cavity were in a similar condition of ulceration to the external parts. The granulations over all the ulcers were unhealthy, and showed no signs of healing except at the parts most distant to the spreading edge. Here such attempts were very feeble. There was very much discharge of a thin, purulent character. So much collected in the naso-pharynx that the patient almost choked every morning before he could get rid of this.

The general condition was poor and the man most miserable.

On March 25th, 20 grs. urea t.d.s. ex Aq. Menth. Pip. 1 oz. were prescribed.

April 25th. Granulations much more healthy, edges distal to spreading edge healing. Discharge very much less. Spreading edges not so raised. 30 grs. urea t.d.s. prescribed.

April 30th. Ulcers quite healthy. Edges less raised. Healing up rapidly from parts first affected. 40 grs. urea t.d.s. prescribed and Maltine ½ oz. t.d.s.

May 7th. Edges of ulcers flat. Line of healing epithelium from these. 50 grs. urea prescribed.

May 27th. Ulcers nearly all healed. Little nodules left on temples and on upper lip.

June 4th. 1 drachm urea t.d.s. prescribed.

June 11th. Nodules on temples disappeared, that on lip disappearing.

At present time the forehead is practically healed, a small sore on the lip,

Digitized by Google

owing to the local application not suiting it, formed, and has not yet healed again.

The local application during the whole treatment was not changed, nor was patient's diet or way of living. His diet was chiefly potatoes, and had been for years. His mode of living was to keep in one room all day, and come out to sell papers in the evening.

Case 2.

Man, aged thirty. Father died of phthisis. Nine years ago lupus commenced on right side of face, and gradually spread round to under chin, healing behind as it travelled, except at junction of lobule of ear with face.

Four years ago left side of nose and face was attacked.

The patient also developed strumous dactylitis in second finger of right hand four years ago, this eventually healed with deformity, leaving a tuberculous dermatitis on back of right hand.

June 24th. Urea grs. 30 t.d.s. was prescribed. The patches were then all raised and nodular and covered with scales and scabs, under which, on removal, was the jelly-like granulation tissue.

The most marked patch was on left side of nose, as big as half-a-crown.

July 2nd. Very great improvement. The raised areas are level at the circumferences with surrounding skin, and are less red. Sensation in them has improved. The scabs are now limited to central parts. Urea continued.

July 9th. The skin, which was stiff and hard where it was affected, is getting pliable and softer. The patches of lupus are melting away. Patient is still under treatment.

Case 3.

A man, *aet.* twenty-two. Glands both groins began to swell a month ago. No apparent cause found. One gland on left, softening in centre, opened, and typical tuberculous caseous gland substance scraped out. Other glands enlarged, and on the right, one softened. Urea grs. 30 t.d.s., increased gradually to grs. 40 t.d.s. The patient, who looked poor and thin, rapidly improved, and the glands did remarkably well; the one that was first scraped out healed in three weeks, the other enlarged glands undergoing resolution. The man was at work in three weeks. Diet and general conditions fairly good.

Case 4.

Woman, *aet.* thirty, married. Family history of phthisis. Glands enlarged for fifteen years left side of neck. Operated on four years ago, and again by myself six months ago, when I removed glands from base of skull to clavicle. Recurred two months ago, just under angle of jaw, and when put on urea, there was a large mass of glands in this situation pushing out the skin and looking as if they must come through. After urea grs. 35 t.d.s. for six weeks, these glands have almost disappeared, and the woman is better in every respect. Still under treatment. Diet and conditions fair.

Digitized by Google

Case 5.

Boy, *aet.* fifteen. History of phthisis.

History of two months' pain and weakness in right elbow. The joint was almost fixed at a right angle, and very painful. All the hollows were filled with typical soft tuberculous granulation tissue in the synovial membrane. Wasting of forearm half inch. No history injury.

July 2nd. After urea gr. xx t.d.s., and rest on a splint for ten days, patient was much improved. Could flex forearm on arm, so that fingers could touch shoulder. Extension not so good. Pronation and supination nearly perfect. All these movements done without pain.

The swelling about the elbow was very much diminished.

July 9th. Swelling about elbow has disappeared altogether. Movements as before, but extension from full flexion to within twenty-five degrees of full extension. Still under treatment. Diet and conditions fair. Urea 1·4 per cent. Average total quantity urine, 36 ozs.

Case 6.

Girl, *aet.* eighteen. History of phthisis on mother's side. Lupus commenced two years ago in nostrils. Glands enlarged in both submaxillary regions and broke leaving ulcers. In the left nostril only the orifice could be seen, owing to granulations of jelly-like material and swelling of the parts. There was a typical patch of lupus about the left part of the upper lip and cheek.

July 9. After a month with urea grs. xxx t.d.s. the left nostril can be examined with ease and the septum seen to be occupied by a healthy granulating ulcer. The lupus on the lip and cheek have all but faded away, and the tuberculous ulcers in the submaxillary regions have healed. Patient under treatment. Diet and general conditions poor.

Case 7.

Patient, *aet.* twenty-two. Many enlarged glands on right side of neck, together with large tuberculous abscess under sterno mastoid from the softening of one. The abscess was opened, washed out and stitched up, no drain being used. In six weeks' time, after an average dose of 30 grs. urea three times a day, the whole of these enlarged glands had entirely disappeared. There was a history of phthisis in this case, but diet and general conditions were very good.

In all other tuberculous cases in which I have given urea myself I am having, or have had, good results.

Not a single untoward symptom has arisen; on the contrary, the action has evidently been in many cases that of a nervous tonic.

In conclusion, I will draw attention to the fact that Dr. Harper has already laid stress in the *Lancet* on the facts that the tubercle bacillus in the laboratory

Digitized by Google

grows best on media that are of low nitrogenous value, and that in the lower animals it attacks the herbivorous rather than the carnivorous.

The cow is an interesting animal, from this point of view, as it is the animal more often attacked by tuberculosis than any other. Here we have a large amount of energy expended in procuring a sufficiency of nitrogen from herbs and a large loss daily in proportion in the casein of milk. This process of extraction of nitrogen goes on far beyond the ordinary period of lactation. It would seem that there may well be a relationship between nitrogenous absorption, assimilation and elimination, and the tubercle bacillus in this case.

I sincerely hope that elaborate experiments will be made with urea in the near future, and that it will be widely tried in all forms of tuberculosis.

Personally I believe that Dr. Harper's discovery will lead to a revolution in the treatment of this disease, and that many cases which have been considered hopeless in the past will be cured in the future.

TREATMENT OF ANOREXIA IN THE TUBERCULOUS BY PERSODINE (PERSULPHATE OF SODA).

By Dr. J. HOBBS,

Professor and Lecturer, Faculty of Medicine; Physician in the Hospital of Bordeaux.

We all know that the condition of a tuberculous patient is measured by that of his stomach. The subject of tuberculosis who can take nourishment is in a good condition to struggle against the bacillus, and to hope for cure; unfortunately, it frequently happens that from the time of the first symptoms, and even when the pulmonary symptoms are little advanced, there is an absolute anorexia. In spite of the advice of his physician, who orders him abundant feeding, the tuberculous patient makes every effort, and does not succeed, in eating. It is in vain that one bitter after another is tried; they all fail, emaciation proceeds apace, and cachexia little by little is established. Many drugs besides bitters have been tried with a view to combating the anorexia, and quite lately chemistry has supplied us with a new product destined to restore the function of nutrition in the tuberculous, and call oxydation into activity. This product is persulphate of soda, the first preparation of which under the name of persodine is due to MM. A. and L. Lemière, of Lyon.

These workers have at the same time produced mineral persulphates of potassium, sodium, ammonium, barium, and lead, and some organic persulphates, the chief of which are the persulphates of cocaine and quinine. These very instable salts have been studied from different points of view by MM. J. Nicolar, Garel, Rigot (of Lyons).

In this study we shall confine ourselves solely to the part they play in nutrition, and we shall direct our attention to one salt alone, persodine

Digitized by Google

(persulphate of soda). Leaving also on one side those cases in which we have prescribed this drug for chorea, anæmia, neurasthenia, etc., we shall concentrate our attention on one particular instance: anorexia in the tuberculous treated by persodine.

The cases we have observed are six in number. Summing them up very briefly, we class them in the following manner: A case of doubtful pulmonary tuberculosis in a girl of twelve years—doubtful in the sense of our not finding bacilli in the sputa, in spite of positive stethoscopic signs. Three cases of pulmonary tuberculosis in the first stage in two children of five and eight respectively, and in an adult male of twenty-four years, the latter with fever. Tuberculosis in the second stage in an adult male of forty-nine, also the subject of fever. Tuberculosis in the third stage in a male adult of twenty-eight, with great increase of temperature.

These six patients were troubled with an anorexia more or less severe, the children principally suffering. We have given persodine to all of them an hour before meals, in a little water—a teaspoonful at a time for children; for adults, a tablespoonful.

From the second day, in the case of the child attacked with doubtful tuberculosis, and in the case of one of those that had reached the first stage, the appetite returned; and from the fourth day it was regained in the cases of the other patients.

In all of them it was shown by a sensation of emptiness at a level with the pit of the stomach, together with a real craving for nourishment. On the fifth day the parents of one of our little patients told us that the child ate greedily.

All our patients increased in weight: the little girl attacked with doubtful tuberculosis gained 4 kilogrammes in twenty days! the other two from 3 to 3½ lbs. Two of our adult patients noticed their weight increase by 60 to 62, and by 70 to 71·500 kilogrammes respectively in twenty-five days.

Even our patient in the third stage derived some benefit from it. The emaciation, which had been progressive, was arrested; there was an increase in weight of 500 grammes, which unhappily at that point went no farther. To sum up we may say that all our patients have increased in weight from the use of persulphate of soda.

In the case of one patient we were able to make a full analysis of urine; dephosphatisation was arrested.

In spite of the rather large doses—two tablespoonsful a day—none of our patients suffered from diarrhœa.

We conclude, therefore, that persulphate of soda stimulates nutrition, and that it is effectual in combating anorexia in tuberculous patients.

Digitized by Google

SUR UN NOUVEAU MODE D'EXCITATION DE LA PEAU DANS LE TRAITEMENT DE LA TUBERCULOSE PULMONAIRE—LE MASSAGE PNEUMATIQUE.

PAR M. LE DR. CH. BREUILLARD, *St. Honoré-les-Bains, France.*

IL est définitivement admis aujourd'hui que la cure hygiéno-diététique doit être la base du traitement de la tuberculose pulmonaire, et la triade, comportant le repos, l'aération continue, et la suralimentation, constitue essentiellement cette cure. Mais il s'en faut de beaucoup que l'on soit d'accord sur la conduite et la dosage de ce traitement, auquel, en dehors de diverses médications, doivent être adjointes certaines autres pratiques hygiéniques plus ou moins importantes, qui rentrent pour la plupart dans le domaine des agents physiques et naturels.

Parmi ces pratiques hygiéniques, aucune ne paraît plus nécessaire que la mise en œuvre des moyens ayant pour but d'activer les fonctions de la peau; et cette nécessité est d'autant plus urgente, qu'aucun des éléments de la triade ne semble pouvoir être institué d'une façon sérieuse et donner des résultats sensibles, si le fonctionnement intégral de la peau n'est pas sollicité par une gymnastique méthodique et disciplinée.

Sans ce fonctionnement, le traitement par le repos donne lieu à des inconvénients graves du côté de la circulation et de la nutrition, l'aération continue est loin de produire tous ses bons effets, en raison de miopragie respiratoire, et la suralimentation devient dans de très nombreux cas une pratique illusoire qui ne donne pas les bénéfices que l'on en attend.

Aussi le principal but de cette communication est de montrer que l'excitation cutanée chez les phtisiques doit aller de pair avec les trois éléments de la triade, et former avec eux une tétrade, si l'on me permet ce mot.

Sans doute, c'est une habitude assez banale et courante en phtisiothérapie que de prescrire l'excitation cutanée, mais en réalité cette prescription, faite d'une façon vague, est exécutée mollement, sinon complètement délaissée, et cela pour diverses raisons d'ordre théorique et technique qu'il semble utile d'indiquer.

Théoriquement, on considère trop la peau comme un simple émonctoire, et on semble ignorer que c'est avant tout un organe nerveux, dépendant, de par son développement embryonnaire et ses fonctions, du système nerveux central. Si, comme l'a fait très heureusement Brissaud, on compare le neurone à un arbre, les fibres cellulipètes, qui s'épanouissent et se terminent dans la peau, sont les racines de cet arbre, lesquelles transforment les diverses excitations venues du dehors en force nerveuse et la portent au tronc et à la frondaison, qui sont représentés par la cellule et ses prolongements, où elle s'accumule ainsi que dans les neurones d'association, pour se déverser ensuite dans les divers organes selon leur besoins nutritifs et fonctionnels. Telle est actuelle-

Digitized by Google

ment la conception classique du réflexe cutané, dont l'importance s'affirme chaque jour, à la suite des observations et des travaux des neurologistes, qui ont mis en évidence les rapports étroits qui existent entre l'innervation de la peau et celle des organes profonds.

C'est à un médecin anglais *Henry Head* (*Brain*, 1893), que l'on doit les plus belles observations faites à cet égard. Cet auteur a remarqué que dans la plupart des affections viscérales, il existe à la peau des zones d' hyperesthésie prononcée, et à contours bien délimités, en rapport avec le viscère malade. De plus, ces zones affectent la forme segmentaire au lieu de suivre les divisions nerveuses ou radiculaires, et Brissaud a mis ces observations à contribution, à côté de beaucoup d'autres portant sur la trophicité et la motilité, pour baser la théorie de la métamérie spinale qu'il a si brillamment édifiée.

Dans la tuberculose aiguë en particulier, Head a signalé de nombreux points d'hyperesthésie qu'il a fixés par des schémas peut-être trop réguliers, mais qui n'en sont pas moins très intéressants.

Sans doute, on avait observé depuis longtemps des anesthésies, des névralgies, et des amyotrophies sur le thorax des phtisiques ; mais ces symptômes, considérés comme peu importants, n'avaient pas été classés, et on n'en avait pas étudié la pathogénie. En laissant de côté les distributions que ces symptômes peuvent affecter sur les territoires nerveux périphériques, radiculaires ou métamériques, on admet aujourd'hui qu'ils sont fonction de névrites, lesquelles se développent en suivant à rebours la voie de l'arc réflexe.

On pourrait croire que ces névrites périphériques sont le produit de toxines bacillaires, mais il semble impossible d'adopter cette pathogénie, attendu que les symptômes apparaissent à l'origine même des localisations tuberculeuses sur les poumons et avant les moindres signes stéthoscopiques. De telle sorte que les troubles observés du côté de la sensibilité, de la trophicité et de la motilité du thorax constituent un moyen de diagnostic précoce de la tuberculose.

En France, Roussel de St-Etienne a bien étudié ce signe précoce sous le nom de *réflexe inhibitoire*, lequel consiste en un soulèvement imparfait de la clavicule et des côtes, et en une insuffisance d'action du diaphragme du côté malade. D'ailleurs, la radioscopie a permis de constater que dans les cas de tuberculisation unilaterale commençante, le diaphragme descend moins bas que du côté sain, tout en remontant aussi haut. Plus tard, quand la germination des tubercules a progressé d'une façon sensible, et au besoin des deux côtés, on finit par observer ce qui a été décrit sous le nom de *thorax paralytique*.

Ce n'est que longtemps après que les polynévrites observées sur divers territoires périphériques peuvent être attribuées en partie aux toxines bacillaires et aussi à celles des infections secondaires. Par conséquent les symptômes précoces en question sont bien dûs à des troubles de nature réflexe, qui donnent lieu à de véritables névrites. On peut admettre que dès qu'une localisation bacillaire existe sur un point des poumons, les cellules des neurones, dont les prolongements partent de ce point, ont leur nutrition modifiée ou entravée dans la région du nevraxe qui préside à l'innervation du point intéressé, et ce trouble de nutrition se répercute directement par les

Digitized by Google

neurones contigus, ou par les neurones d'association sur les extrémités des fibres cellulipètes qui constituent les nerfs sensibles de la peau. On peut même se demander à ce propos si les doigts hippocratiques, que l'on observe chez certains sujets bien avant les signes de la cachexie consomptive, ne révèlent pas un trouble trophique ayant le même mécanisme pathogénique par voie réflexe.

Quoiqu'il en soit, il est certain qu'une fois ces névrites établies, le rhythme respiratoire est modifié, l'ampliation thoracique est diminuée du côté malade et devient dans ce cas asymétrique. Il se constitue ainsi une miopragie ou insuffisance respiratoire qui favorise les congestions broncho-pulmonaires et, par suite, de nouvelles germinations tuberculeuses.

Il y a donc urgence à sortir de ce cercle vicieux en luttant contre ces névrites secondaires qui, à plusieurs égards, entretiennent l'affection primitive, et il est d'observation traditionnelle que la médication révulsive est la meilleure que l'on connaisse pour les combattre et les faire disparaître.

Mais, quel procédé révulsif faut-il employer? C'est à ce propos qu'il est bon de signaler la deuxième raison d'ordre théorique qui explique le délaissement dans lequel sont tombés les divers modes d'excitation cutanée en phtisiothérapie. Cette raison réside dans la confusion extrême des idées régnantes sur la thérapeutique révulsive. D'ailleurs le mot de révulsion beaucoup trop compréhensif et par conséquent très vague explique par lui-même cette confusion, qui peut aussi dériver en partie des théories physiologiques actuelles sur l'excitation.

On admet en effet que toute excitation portant sur un tronc nerveux ou sur la peau est univoque! Que cette excitation soit de nature thermique, électrique, chimique, ou mécanique, elle agit de la même façon sur les grandes fonctions organiques (respiration, circulation, calorification, nutrition). Les diverses expériences tentées dans ce sens ont fait admettre cette opinion qui semble n'avoir guère pour elle que la vraisemblance, mais qui n'en a pas moins produit un grand trouble dans le choix des divers agents révulsifs, puisque ce choix paraît généralement n'avoir pas d'importance.

Je ne m'attarderai pas à envisager les divers moyens d'excitation et d'irritation cutanées qui ont été employés en phtisiothérapie, car il faudrait passer en revue toute la matière médicale de la révulsion. Je mentionnerai seulement à part l'antique vésicatoire, dont l'action sédative est indéniable vis-à-vis des symptômes aigus, et principalement dans les congestions et les poussées de broncho-pneumonies pérituberculeuses. Mais je me crois obligé ici de comparer, aussi brièvement que possible, ce qui à ma connaissance n'a pas encore été fait, les quatre procédés d'excitation cutanée les plus répandus et que l'on emploie plus ou moins et un peu au hasard, pendant la cure hygiéno-diététique.

Ces procédés sont la cautérisation ponctuée, certaines modalités électriques, l'hydrothérapie, et le massage.

La pratique des pointes de feu multiples et répétées sur le thorax des tuberculeux, à l'aide de ces admirables instruments de chirurgie qui sont le thermo-cautère et le galvano-cautère, est assez délaissée depuis quelques années.

Digitized by Google

On ne peut cependant nier les succès de cette médication, mais on a reconnu que l'action tonique et dynamogénique obtenue dans les premiers temps de l'application s'affaiblit très vite. L'explication de ce résultat semble résider dans le fait qu'à chaque application on détruit d'une façon irrémédiable une immense quantité de terminaisons nerveuses, dermiques, et épidermiques, de sorte qu'après un certain temps la presque totalité de la surface thoracique n'est plus guère qu'une immense cicatrice, superficielle sans doute, mais plus ou moins anesthésiée et, par suite, incapable de donner lieu aux réflexes cutanés normaux dont la nécessité s'impose par dessus tout en pareil cas. J'ai été à même depuis longtemps d'examiner un grand nombre de tuberculeux, dont j'ai dirigé la cure à St. Honoré-les-Bains, et j'ai toujours constaté le résultat que je viens d'énoncer, ce qui semble expliquer et justifier la défaveur croissante qui s'attache à ce procédé d'ailleurs assez barbare.

Certaines modalités électriques ont été proposées et employées pour exciter la peau des phtisiques. On a recours parfois à la faradisation cutanée, qui, dans les cas de névralgies récentes, agit vite et bien. On a employé dans les mêmes circonstances l'étincelle statique, qui a également donné des résultats satisfaisants. Enfin on a proposé tout récemment l'emploi des effluves mono- et bipolaires de haute fréquence sous la forme d'une pluie d'étincelles crépitant sur la peau de toute la surface thoracique. Le professeur Doumer de Lille a fourni des observations assez probantes en faveur de cette méthode.

J'ai eu l'occasion d'examiner plusieurs malades qui étaient soumis à ce dernier traitement dans deux établissements d'électrothérapie à Paris, et il ne m'a pas semblé que les résultats physiologiques et thérapeutiques fussent supérieurs à ceux donnés par la faradisation cutanée ou par l'étincelle statique. Dans tous ces cas, la peau de la surface thoracique, qui n'est le siège d'aucune réaction durable et profonde, peut être réveillée de sa torpeur. Certaines anesthésies, hyperesthésies ou dysesthésies récentes peuvent être promptement améliorées ; mais quand la névrite est définitivement installée avec son cortège de symptômes, elle se montre rebelle aux diverses modalités électriques, aussi bien chez les tuberculeux que chez les autres malades. En un mot l'action sur les centres nerveux provoquée par la sensation douloureuse résultant de ces diverses modalités est purement dynamique et superficielle, et est incapable par elle seule de susciter des modifications statiques et profondes comme celles provoquées par des excitations physiques plus matérielles et plus effectives, telles que la température et certaines manipulations mécaniques, comme la percussion, le pincement, la malaxation, etc.

Déjà Duchenne de Boulogne n'expliquait la cessation de la douleur névralgique à la suite de la faradisation cutanée, que par ce qu'il appelait la *contre-fluxion douloureuse*. Il ne semble pas qu'on puisse trouver aujourd'hui une explication d'un autre ordre. On a invoqué, à la suite des beaux travaux de d'Arsonval sur les courants de haute fréquence, une action spéciale de ces courants sur la nutrition ; mais cette action, évidente sans doute, semble être d'ordre banal et ressemble à celle d'un grand nombre d'autres agents physiques.

Il n'est pas inutile d'avancer à ce propos qu'on a singulièrement abusé

Digitized by Google

de l'action de diverses médications sur la nutrition générale pour tenter d'expliquer leur action spéciale : s'il est certain que tout changement dans les fonctions d'un organe ou d'un appareil implique une modification trophique que l'on est en droit de soupçonner, il s'en faut encore que nous puissions entrevoir les mutations cellulaires inhérentes à tel tissu en particulier, et force est pour le moment de constater les transformations fonctionnelles survenant à la suite d'excitations cutanées, sans pouvoir les expliquer.

C'est le cas maintenant, pour continuer la comparaison commencée, d'envisager l'hydrothérapie, laquelle a ses chauds partisans parmi les phtisiothérapeutes et, en fait, des succès sérieux et éclatants légitiment l'emploi judicieux de cette médication. Des observateurs comme Brehmer de Goerbersdorf, et surtout le Professeur Winternitz de Vienne, ont produit des statistiques fort encourageantes ; mais il s'en faut qu'on puisse doucher sans danger tous les phtisiques. Dans le cours de ma carrière hydrologique j'ai vu de nombreux tuberculeux incapables de toute réaction. J'en ai vu d'autres qui, après avoir paru se trouver admirablement des premières douches, s'affaiblissaient très vite pendant la suite du traitement. En somme, si l'hydrothérapie donne des résultats beaucoup plus marqués que l'électrothérapie, elle semble être, beaucoup plus que cette dernière, une arme à deux tranchants et par suite dangereuse.

C'est qu'il faut considérer dans l'hydrothérapie deux actions bien distinctes : l'action réactionelle, qui exalte les réflexes cutanés et qui seule doit être recherchée en phtisiothérapie, et l'action réfrigérante qui, très utile pour combattre l'hyperthermie, n'est pas de mise chez les tuberculeux chroniques. Or l'action réfrigérante se produit de trois manières : par la soustraction directe du calorique, par l'évaporation de l'eau imbibant l'épiderme, et enfin, par le rayonnement du calorique pendant la réaction, laquelle donne lieu à une congestion active de la peau.

Ce dernier mode de réfrigération est le seul qui semble devoir être recherché et être utile. On peut ajouter qu'il est le seul qui soit physiologique, pour ainsi dire, tandis que les deux autres modes de réfrigération sont purement physiques et par conséquent extra-physiologiques. Par suite, l'organisme a plus à faire pour récupérer cette perte de forces.

Sans entrer dans d'autres détails, il semble permis d'admettre que l'hydrothérapie ne peut être prescrite aux tuberculeux qu'en s'entourant de nombreuses précautions, tout en s'exposant malgré tout à différents risques plus ou moins graves. C'est en somme un procédé facile à employer pour exciter les fonctions nerveuses de la peau, mais qui doit être considéré comme assez grossier et souvent dangereux pour les phtisiques.

J'arrive enfin à parler du massage comme mode d'excitation cutanée chez les tuberculeux ; mais énoncer ce programme, c'est se heurter aussitôt à l'opinion courante qui consiste à admettre que le massage manuel et traditionnel agit directement et principalement sur les muscles et les vaisseaux. Cette opinion, qui, à mon sens, est erronée, est due en grande partie à ce que les manipulations masso-thérapiques ont ce but apparent en raison des pressions, percussions, et effleurages centripètes qui peuvent sans doute en imposer à cet

Digitized by Google

égard ; mais si l'on observe de près l'action physiologique et les résultats thérapeutiques de ces manipulations, on est obligé d'admettre qu'elles agissent *avant tout et toujours* sur les nerfs sensitifs cutanés et sous-cutanés.

Les limites qui me sont imposées par les statuts du *Congrès* ne me permettent pas d'entrer dans tous les détails nécessaires pour démontrer cette manière de voir, que je traite à fond dans une brochure actuellement sous presse.* Qu'il me suffise de dire que, dans ce travail, je présente un grand nombre de faits et d'expériences comparatives qui semblent prouver d'une façon absolue que le massage seul de la peau fait dans le sens centrifuge, et tel qu'il est d'ailleurs pratiqué en Chine et dans tout l'extrême orient, produit des effets semblables, sinon supérieurs, à ceux que donne le massage dit musculaire et centripète, pratiqué en occident.

J'arrive, en prenant pour base d'observation une maladie bien définie et dont le champ d'action s'agrandit chaque jour, la névrite périphérique, à montrer que le massage bien pratiqué peut être considéré comme le véritable spécifique de cette affection, en laissant bien loin en arrière les résultats obtenus par les diverses modalités électrothérapiques, les procédés révulsifs et analgésiques les plus actifs tels que les pulvérisations de chlorure de méthyle, et les meilleures pratiques hydrothérapiques.

En observant de près et en analysant les manipulations multiples et souvent superflues du massage manuel, je crois être arrivé à démontrer que le mécanisme par lequel on obtient des modifications aussi importantes consiste dans la traction et au besoin l'élongation des fibrilles et des filets nerveux cutanés et sous-cutanés. Cette traction et cette élongation sont les moyens les plus puissants que l'on connaisse jusqu'ici pour exalter les réflexes et exhausser l'action trophique des centres médullaires. On peut rapprocher ces effets de ceux décrits récemment par Chipault dans ses travaux sur l'élongation des nerfs pour la cure du mal perforant et des ulcères variqueux et de ceux produits par les tractions rhythmées de la langue si merveilleusement imaginées par Laborde !

De toutes ces considérations, on peut se croire autorisé à conclure que le massage constitue réellement le procédé de choix pour exciter la peau et en particulier celle des tuberculeux.

Mais le massage bien pratiqué est chose beaucoup trop difficile et trop absorbante pour le médecin, et reste forcément une méthode d'exception et de luxe, à laquelle on ne peut pas songer dans le traitement de la grande majorité des phtisiques. En outre, les résultats obtenus sont *artistiques*, c'est-à-dire inconstants et variables selon les aptitudes spéciales des professionnels. De sorte qu'en adoptant cette médication, on s'expose fatalement à des mécomptes et à des impossibilités de tous genres ; telles sont les principales causes de l'abandon presque absolu des pratiques massothérapiques, dans le traitement de la tuberculose pulmonaire.

---

* " Un nouveau mode d'excitation cutanée : Le massage pneumatique." Par le Dr. Breuillard, de St. Honoré-les-Bains ; chez Berthier, librairie médicale, 104, Boulevard St. Germain, Paris, 1902.

Digitized by Google

C'est après avoir reconnu ces difficultés techniques à peu près insurmontables, qu'à la suite des vues exposées plus haut sur le mécanisme thérapeutique du massage manuel, j'ai imaginé le massage pneumatique, qui semble destiné à remplacer ce dernier dans la grande majorité des cas.

L'appareil est composé essentiellement d'un bloc de caoutchouc creux, qui porte le nom *d'ampoule cursive*, et qui peut être facilement pris à la main, à la façon d'une brosse. Dans la cavité ovoïde de cette ampoule molle et flexible aboutit un tube, également en caoutchouc, plus ou moins long et branché sur un appareil à vide.

Cet appareil à vide tout nouveau est constitué par un éjecteur à eau, fixé sur un socle-réservoir contenant un litre d'eau environ, et muni d'une petite pompe double aspirante et foulante mue par les pieds. C'est toujours la même eau qui, refoulée par les pieds, au moyen de pédales, passe par l'éjecteur en faisant le vide et revient dans le socle-réservoir, où elle est reprise à nouveau. Cette nouvelle machine pneumatique est indéréglable !

Aussitôt que l'appareil est mis en train par l'opérateur, le vide est produit dans l'ampoule, et si cette dernière est appliquée sur un point quelconque du corps d'un sujet, la peau est aussitôt frappée et serrée dans la cavité. C'est alors qu'en promenant cette ampoule sur le corps à la façon d'un fer à repasser, on produit une traction de la peau dont la force varie de 10 à 20 kilos, selon le vide produit et une rougeur plus ou moins vive se manifeste.

Le vide peut varier entre 30 et 60 centim. de mercure, et si la peau est prise à la nuque, par exemple, on peut ainsi la tirer jusqu'au talon.

L'action mécanique du massage pneumatique peut être résumée en quatre mots : succion, friction, expression et traction de la peau. C'est cette dernière qui est de beaucoup la principale, en produisant le tiraillement et au besoin l'élongation des filets nerveux cutanés et sous-cutanés.

La sensation produite est une douleur spéciale, assez vive, mais très supportable, puisqu'elle cesse instantanément après l'application de l'ampoule. Elle est comparable à celle de la faradisation cutanée moyenne, mais n'est pas agaçante comme cette dernière. Cette douleur est également moins désagréable que le froid pénétrant et horripilant de l'hydrothérapie, et cependant, après une séance de trois à cinq minutes de massage pneumatique généralisé, une sensation de chaleur et d'excitabilité musculaire se manifeste, qui a beaucoup de rapports avec ce que l'on éprouve pendant la réaction qui suit une douche bien supportée. C'est bien d'ailleurs une réaction plus franche, plus sûre, et qui dure plus longtemps que la réaction hydrothérapique et cela sans le choc bien connu de la douche froide, et sans encourir la moindre chance de refroidissement.

Ainsi qu'on l'a déjà vu, aucune réaction semblable ne se produit à la suite de l'application de diverses modalités électriques. Rien d'analogue n'est pas plus remarqué, ni ressenti, à la suite d'une application vibrothérapique, ou de ce que l'on a appelé le massage vibratoire. Cette dernière modalité mécanique dont l'action thérapeutique semble avoir été très exagérée, ne produit guère que des effets analgésiques momentanés, lesquels sont appré-

Digitized by Google

ciables dans certains cas, mais ne sont pas comparables aux mêmes effets durables produits par le massage pneumatique.

Les effets thérapeutiques du massage pneumatique sont naturellement nombreux et importants, et dérivent de son action particulièrement élective sur la sensibilité cutanée; mais je n'ai à considerer ici que la mise en valeur ou l'influence de cette sensibilité dans le traitement de la tuberculose pulmonaire.

Dès les premières séances, le massage étant appliqué principalement sur le tronc et les bras, l'oppression est soulagée, la toux est diminuée, et l'expectoration est rendue plus facile. Cette sédation remarquable est le fait de l'exaltation des reflexes cutanés qui donnent lieu à l'excitabilité des muscles thoraciques, et par suite à une ampliation bronchique plus large.

Les douleurs intercostales cessent, l'appétit augmente, et on assiste à ce remontement général bien connu que l'on observe d'ordinaire, à la suite des applications hydrothérapiques les mieux supportées. En outre, certains medicaments, comme la créosote et l'huile de morue, qui n'étaient pas supportés par de nombreux malades, sont admirablement tolérés.

Tous ces bons résultats ne peuvent être expliqués que par une action tonique et dynamogénique de premier ordre, de laquelle bénéficie le malade restant au lit, où il respire plus largement et où son appétit augmente.

Comme on le voit, la triade hygiéno-diététique est grandement facilitée, et cela sans l'emploi de révulsifs et d'autres modificateurs physiques, difficiles à appliquer, offrant certains inconvénients et au besoin dangereux.

Il me reste à signaler les contre-indications du massage pneumatique, qui en principe se résument en une seule: la fièvre consomptive. Cette médication n'est pas applicable quand la fièvre est continue, que les lésions pulmonaires sont étendues, et que la cachexie consomptive est nettement établie. Dans ce cas, l'excitation des réflexes cutanés augmente la fièvre et les forces diminuent promptement. Il n'y a donc pas à insister pour montrer que l'abstention est absolument indiquée en pareil cas. A ce propos, je saisis l'occasion qui m'est offerte de ne pas accepter une partie des conclusions de la communication faite par M. Alb. Robin, tant en son nom qu'en celui de M. Binet, au cours de l'avant-dernière séance du Congrès, sur "*La prophylaxie de la tuberculose pulmonaire par la connaissance de son terrain.*" Je laisse entièrement de côté la question de l'exagération des échanges respiratoires à toutes les périodes de la maladie, ce qui oblige d'admettre que tout tuberculeux est un consomptif, car les auteurs n'ont pas encore fait connaître la technique dont ils ont fait usage et surtout les conditions expérimentales dans lesquelles ils ont opéré, pour ne retenir et n'envisager ici que la conclusion ayant trait à la prophylaxie et à la thérapeutique.

"Le terme vague de déchéance organique héréditaire ou acquise, disent nos collègues, dont on s'est servi jusqu'à présent pour définir le terrain de la phtisie, ne comporte aucune indication prophylactique ou thérapeutique. On dit aux alcooliques de ne plus boire, aux surmenés de se reposer, et aux descendants de tuberculeux de se tonifier; mais le propre des médications

Digitized by Google

dites toniques est justement de stimuler les échanges organiques, qui sont déjà chez ces héréditaires en état de suractivité ; au contraire, l'analyse des échanges respiratoires révèle une condition commune aux états de prédisposition et dont la connaissance comporte une nette indication thérapeutique qui est de restreindre le pouvoir de l'organisme à fixer trop d'oxygène et à faire trop d'acide carbonique. Il ne s'agit donc plus que de rechercher quelles sont les médications et les médicaments capables de réaliser cette indication."

Si l'on met à part le jugement sur les conseils donnés aux intempérants de ne plus boire et aux fatigués de se reposer, conseils surannés sans doute, mais qui paraissent être encore de quelque utilité, il semble difficile d'admettre cette proposition toute gratuite que " le propre des médications toniques est justement de stimuler les échanges organiques, qui sont déjà chez ces héréditaires (de tuberculeux) en état de suractivité." Sans doute, il est possible et même certain que dans des cas déterminés, la médication tonique stimule l'activité des échanges ; mais il est indéniable que dans d'autres cas non moins nets, la même médication restreint cette activité ; de sorte que la réalité des faits semble montrer que la médication tonique a pour principale action de *régulariser* les phénomènes nutritifs.

Cette conception du mode d'action des toniques paraît s'accorder avec les données physiologiques de dynamogénie d'une part, et d'interférence ou d'inhibition de l'autre, consacrant le fait qu'une excitation déterminée exalte les fonctions d'un organe au repos et arrête les functions du même organe en activité.

Il est donc vraiment difficile d'admettre la dichotomie formulée depuis longtemps par M. Robin et qui consiste à ranger les divers médicaments et médications en deux catégories, dont l'une comprend ceux qui apaisent les échanges et l'autre ceux qui les accroissent.

La thérapeutique traditionnelle de la tuberculose pulmonaire a toujours employé avec un succès relatif, mais évident et constant, les divers agents de la médication tonique. Nul doute qu'en continuant à mettre en œuvre cette médication, mais avec plus d'opportunité et de mesure, et en faisant un choix plus judicieux des agents employés, on n'obtienne des résultats plus accentués et plus nombreux. C'est la voie dans laquelle on est entré depuis quelque temps en instituant la cure hygiéno-diététique, qui n'est que la première étape du chemin à parcourir.

Or la cure hygiéno-diététique est d'essence nettement tonique ; par suite, il semble douteux que l'analyse des échanges respiratoires, qui est sans doute intéressante au point de vue bio-chimique, soit appelée à transformer la thérapeutique de la tuberculose pulmonaire, dans le sens indiqué par les auteurs de la communication en question.

En somme, je crois pouvoir maintenir d'une façon générale les contre-indications que je viens d'énoncer, en ce qui concerne l'emploi du massage pneumatique dans le traitement de la tuberculose pulmonaire, en ajoutant qu'il est impossible de préciser ici certaines d'entre elles qui résultent plutôt de l'état général que de l'état local, et que le médecin traitant est seul dans la situation d'apprécier.

Digitized by Google

Pour en finir avec le massage pneumatique, il est bon de spécifier que dans certains cas douteux, où la fièvre n'est pas absolument continue, et quand il reste chez les malades une certaine résistance, il n'y a aucun danger à essayer des applications légères et ne portant au début que sur une partie de la surface thoracique. En procédant de la sorte on voit très vite si le sujet est en mesure de supporter la médication.

Conclusions.

Il a été constaté que l'excitation de la peau constitue une médication de premier ordre dans le traitement de la tuberculose pulmonaire, mais qu'en pratique les procédés donnant lieu à cette excitation sont beaucoup trop délaissés. Cet abandon est dû à des raisons d'ordre théorique et technique que l'on peut résumer en quelques mots : connaissance imparfaite des fonctions nerveuses de la peau et des altérations de ces fonctions chez les tuberculeux ; confusion extrême des idées régnantes sur la thérapeutique révulsive, dont les procédés ne sont pas encore classés selon leur importance et sont appliqués au hasard ; enfin, difficultés d'application et dangers de certains procédés d'ailleurs actifs.

Après avoir comparé entre eux les quatre grands moyens d'excitation cutanée les plus en vogue : la cautérisation ponctuée, certaines modalités électriques, l'hydrothérapie, et le massage, j'ai cru devoir donner la préférence au massage, qui donne lieu à l'excitation cutanée la plus complète, en raison de la traction des filets nerveux cutanés et sous-cutanés, qui ne peut être produite que par cette manipulation, laquelle a une action beaucoup plus sûre et exempte de complications fâcheuses.

Mais le massage bien appliqué est trop absorbant pour le médecin, et reste forcément une méthode d'exception et de luxe. Les résultats obtenus sont artistiques, c'est-à-dire inconstants et variables, selon les aptitudes spéciales des opérateurs.

C'est en me basant sur ces considérations que je propose de remplacer le massage manuel par le massage pneumatique, que j'ai imaginé et qui vient d'être décrit.

En somme, le massage pneumatique, très facile à employer, synthétise les actions de la cautérisation ponctuée, de plusieurs modalités électriques, de l'hydrothérapie et du massage manuel. C'est une nouvelle note à ajouter à la gamme des agents physiques et naturels, note dont le rôle semble très important dans le traitement de la tuberculose pulmonaire chronique.

En dernier lieu j'insiste sur la commodité et la facilité d'application du massage pneumatique, que tout médecin, aussi bien que toute personne un peu adroite, peut pratiquer en quelques minutes, sans fatigue et sans la moindre connaissance de la technique compliquée, délicate, et absorbante du massage manuel traditionnel.

Digitized by Google

TREATMENT OF TUBERCULOSIS BY HIGH-FREQUENCY CURRENTS.

By Dr. J. A. RIVIERE, *Paris.*

Since the memorable experiments of Professor d'Arsonval and Dr. Charrin with regard to the effects produced by high-frequency currents upon bacilli and their toxines, I have been fully persuaded that tubercular patients would derive great benefit from these agents. Since then I have had the opportunity of treating many cases of pulmonary tuberculosis by means of the grand solenoid of d'Arsonval, and the effluves of Oudin's resonator. The patients were submitted to a treatment of twenty minutes' duration every second day. I have invariably remarked, the same as Dr. Doumer and Dr. Oudin, that patients undergoing the treatment have felt their general health improve from day to day, and in certain cases the physical signs of the pulmonary lesions have completely cleared up. The bacteriological examinations also showed that the number of bacilli were fewer in the expectorations. It is to avoid auto-intoxication that I have always advised my patients to take a small dose of calomel now and then; it is quite probable that, at the same time as calomel disinfects the digestive tube, it favours by its cholepoietic action the elimination of the refuse of the organism. This marvellous medicament also very probably exercises a bactericidal action in the remotest parts of the organism. I ought to add that I have always forbidden raw meat, which, in my opinion, merely intoxicates the patient and induces albuminuria. I consider it can only be taken with advantage by carnivora.

I alternated my high-frequency séances with applications of Röntgen rays and inhalations of pure ozone. It seems to me advisable to discontinue treatment during congestive or febrile exacerbations. Electric treatment appears to be most successful in chronic cases.

I now wish to call attention to some convincing results which I have obtained in localised tuberculosis. The first was the case of a patient, aged fifty-five, suffering from a tubercular ulcer situated two centimetres below the middle portion of the left clavicle. It was of an elongated shape, and measured three centimetres in its long diameter, and two centimetres across; it was a centimetre and a half deep, and filled with cheesy-looking purulent matter of a yellowish-green colour; the edges were somewhat indurated, and two of the axillary glands were as large as a pigeon's egg. It had commenced by a painless infiltration of the skin, which, after having taken on a nodular appearance, broke down and ulcerated.

I commenced my applications of the effluves of Oudin's resonator; the ulcer became less painful; instead of being purulent the discharge became serous; the pallid appearance of the base of the ulcer was succeeded by copious red granulations, and in less than a month it completely healed. It was covered with a white membrane, soft and fine in appearance; this process

Digitized by Google

of healing presented a great contrast to what had occurred on the opposite side three years previously in the case of an ulcer of the same nature, which required a whole year's treatment by means of iodoform, ignipuncture, and scraping before it healed, and even then had left a hard-looking cicatrix (kéloïdienne). The daughter of this patient, affected with pulmonary tuberculosis in the first stage, recovered under the combined influence of high frequency of the grand solenoid d'Arsonval and Röntgen rays. Her age was thirty, and her treatment lasted three months.

Three years ago I had under my care a young man suffering from arthritis of the knee of a suspicious nature. The epiphyses were first attacked, and the joint was much enlarged; there was thickening of the articular surfaces of the bones; the peri-articular structures were swollen; the limb was a little bent upon the thigh, and there was pain from time to time. After eighty sittings of localised high frequency, the articulation had recovered its functions, the knee was diminished in size, the swelling of the soft parts, as well as the pain, had completely disappeared; the general health seemed excellent. The patient left for the country. Two months afterwards he wrote to me saying that his health had been completely re-established, but he attributed his cure to the country air. I am persuaded that this patient was suffering at the beginning from tubercular arthritis. The applications were made by means of one or two dampened plates attached to one or both extremities of the small solenoid.

But the most convincing case of the favourable action of high-frequency currents in localised tuberculosis is the following:—

Mr. X., a Scotch gentleman of gigantic stature, was sent to me by a confrère a year ago. He had been operated upon ten months before for tubercular glands of the neck. The disease had clearly broken out again. He had five cervical glands rather smaller than a hen's egg, and there were four fistulous openings freely discharging. I gave him a daily application of long effluves from Oudin's resonator of five or six minutes' duration. From the following day the discharge was changed in character. It became serous instead of purulent, and at the end of the tenth application the fistulas had dried up. The swelling of the neck steadily disappeared, and on the thirtieth application, at the end of a month's treatment, there remained only one sublingual gland enlarged; it was the size of a pigeon's egg. Business obliged the patient to discontinue treatment for a month. He then came back to me with a fistulous discharge which seemed to proceed from the gland in question. After four fresh applications of the effluves the fistula dried up and the gland of the neck considerably diminished in size, when the patient left for the country. On his return I hope to effect a complete cure. This patient, like all the others who have been similarly treated, always told me that under the influence of high-frequency currents his strength increased and sleep and appetite were equally improved.

Since the interesting communication of Dr. Doumer with reference to the cure of anal fissure and fistula, I have had the opportunity of trying the procedure in such cases on six occasions. The cases of fissure got quickly well;

Digitized by Google

two fistulas disappeared after a treatment of one or two months. In the first instance I introduced a small stylet into the fistulous track, this stylet being attached to Oudin's resonator. After four applications of this kind, I used a glass cone filled with metallic filings, and attached to the resonator (condensing electrode). Two other patients who suffered from complete fistula did not end the treatment. But in every case the purulent and fœtid discharge of the first day gave place to a comparatively slight serous discharge with a less offensive odour; the pain rapidly disappeared. In the last two cases, to accelerate the treatment, I had also used creosoted suppositories.

I might add the case of an English lady who had an osseous fistula in the heel. At the beginning of the treatment radiography had shown a small opaque cone-shaped excrescence half a centimetre in length at the lower end of the os calcis. After three weeks' treatment by localised high frequency the fistulous opening had dried up, pain had disappeared, and walking was easy. Three radiographs taken at different intervals show that the osseous changes had gradually diminished until they had completely disappeared.

Conclusions.

It follows from the foregoing that high-frequency currents exercise a certain curative action upon tuberculosis (pulmonary and localised). The microbe cannot resist the repeated application of these currents; its reproductive power and the virulence of its toxines become attenuated, a fact which had already been proved by the laboratory experiments of Professor d'Arsonval. It appears that, at the same time, the human organism under the influence of these currents gets stronger, its enemy, the microbe, grows weaker, and is killed by their actinic rays when large bipolar effluviations are used. The acceleration that these currents bring to the circulation of the blood and lymph favour the osmotic action. This allows the organism to eliminate the refuse and the toxines that invade and infect it. Thus cleansed, the organism is in good condition to struggle against the microbe. It is also probable that under the influence of this form of electrisation the beneficial action of the phagocytes increases in activity. It thus appears evident that in these conditions the attacked organism recovers its strength, and, aided by phagocytes, finally prevails against the bacillus, whose vitality the electric concussions had already imperilled. It is, therefore, sufficient to place the patient in the best conditions of food and hygiene to avoid the return of the disease. It is in this way that some of my patients sent south or in pure air after the treatment have completely recovered. I regret that I have never had the opportunity of treating true white swelling, or Pott's disease. I am under the impression that these two localisations ought to be materially influenced for the better by high-frequency currents.

Digitized by Google

THE TREATMENT OF PULMONARY TUBERCULOSIS BY MEANS OF ELECTRICAL CURRENTS OF HIGH FREQUENCY AND HIGH POTENTIAL.

By CHISHOLM WILLIAMS, F.R.C.S. Ed., etc.,
Surgeon Out-Patients, City Orthopædic Hospital, London, E.C.

We may safely state that there is no medical disease that has had such attention devoted to it by the medical profession of recent years as that of pulmonary tuberculosis. The "open-air" treatment in sanatoria is undoubtedly effective in a great number of the slighter cases, but in the present day "the sanatorium" is beyond the means of the vast majority of sufferers.

Another method is by means of electrical currents of H.F. and H.P., which on the Continent has had many able advocates, and the results have been sufficiently striking to induce the profession in this country to give it a thorough trial. With this object in view I have treated a number of phthisical patients, and from forty-three consecutive severe cases can formulate a favourable opinion of the method.

The patients were picked for the severity of their symptoms, by other medical men. We believe the currents act in the following manner. Firstly, on the tubercle bacilli themselves, by making them pursue the same course as if they were under the "X" rays. According to the experiments of Dr. Norris Wolfenden and Dr. Forbes Ross, the bacilli rapidly increase in numbers, and have a tendency to form clumps, then get small in numbers and shape, and take the microscopical stains very readily, but are pale in colour.

Quite recently Drs. Lagriffoul and Denoyés, of the Montpellier Faculty of Medicine, have published a further series of experiments on the action of "high frequency" on tuberculous guinea-pigs, and have proved that an actual inflammation is produced around the pulmonary foci, that finally this abates and leaves the lung clear of the bacilli. M. Dimitriewski, of the University of Tomsk, has been working with various toxins with a like result—the toxins losing their power and becoming innocuous.

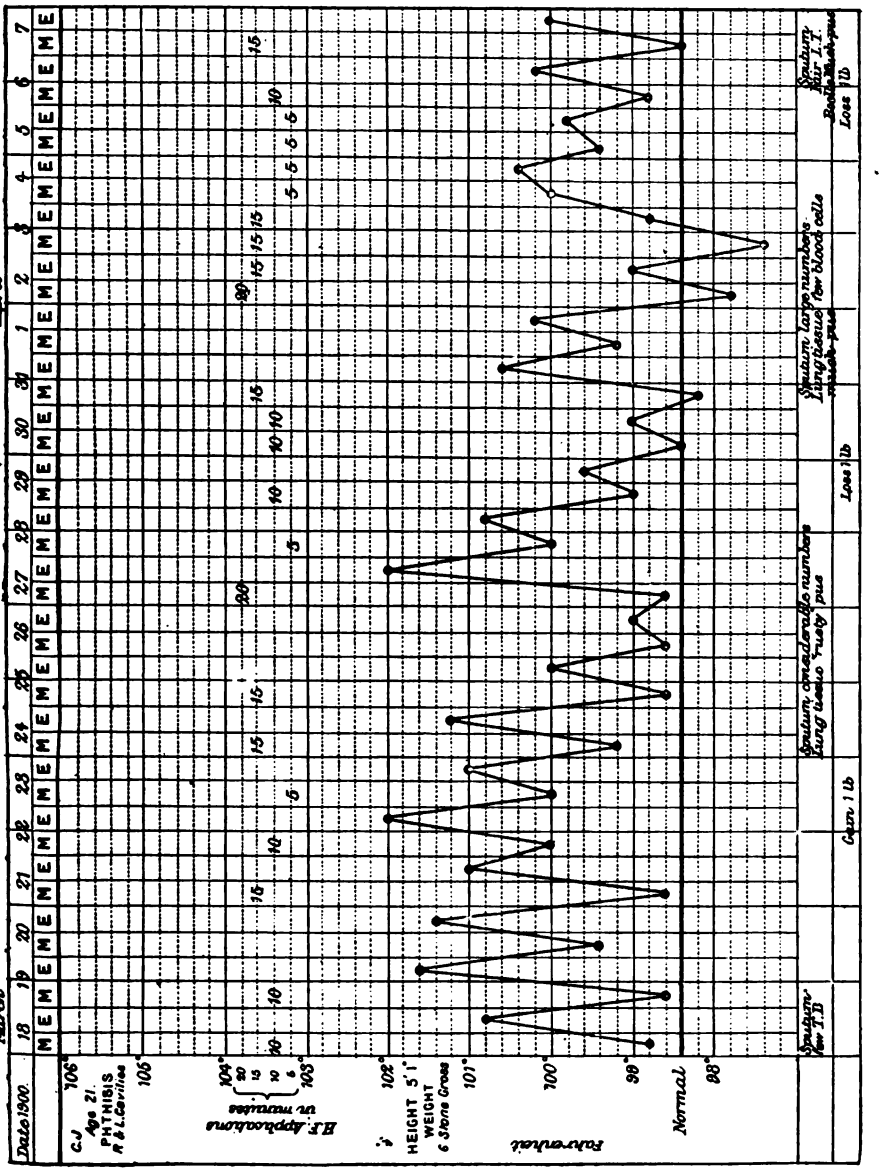
Secondly, the effects of the currents of high frequency, etc., on the individual cells of the body. We judge this by the appetite and digestive powers increasing, and the patient's gain in weight. The general improvement of the body cells probably makes them more resistant to the inroads of the tubercle bacilli; but whether the lowering of the tubercle's vitality, or a raising of the body cells' resisting power, or a combination of both is at work, for our purpose, matters little. In every case one noted that the temperature was the first thing affected, presuming that the daily variation had been of about two degrees between the evening rise and morning fall; either after the first application, or at most after the third, the evening rise was higher and the morning fall much less. On examining the affected area we find the physical signs at first increased, thus more coarse rales, of louder and of

Digitized by Google

greater number, could be found, the expectoration becomes greater, and the cough more frequent and easier. After a few applications, especially local ones, the patient often complains of pain, or an uncomfortable feeling over the affected part; this, as a rule, passes off after a couple of weeks of treatment, and is never severe if we pay due attention to the length of time and number of the applications. A slight amount of pain over the affected area in severe cases is often noted. The tubercle bacilli, which are usually present in fair numbers, quickly begin to increase, and after a few applications are greatly increased. They soon, however, form clumps and get misshapen, short and stumpy, and generally curved, and take the stain far more readily than before; after a time they begin to decrease in numbers, and later on, when the patient is obviously getting better in every respect, they may cease entirely, and may reappear in the sputum after weeks of absence. The physical signs generally persist for a considerable time after the patient has regained his normal weight (for height), and all signs and symptoms are absent or in abeyance. The consistency of the sputum is interesting. At first, besides bacilli, we get very quickly after the application of the H.F. currents, lung tissue and a few blood cells, both of which may greatly increase, the former showing the alveolar arrangement, and the latter being in great numbers, producing the typical "rusty" sputum of pneumonia. Pus is often found; later the sputum clears, leaving only a few bacilli and mucus. In eight cases I have noted semi-calcareous nodules expectorated. The sputum, which at first is generally muco-purulent, and may be offensive, loses its offensiveness and becomes less coloured. Generally in the course, during a varying time, extending from a few weeks to a month or more, it is very heavy and sinks. As the patient progresses it becomes lighter in weight and whiter in colour, far more serous, and easy to "get out." Tubercular laryngitis seems to be particularly amenable to the effect of the H.F. currents, as do tubercular skin affections, lupus, etc. The diarrhœa which I met in the majority of my cases was seemingly easily corrected by the treatment, as the digestion improving, the intestines were less worried by the irritating waste products.

When the temperature has been raised by the treatment the patient, of course, may feel rather worse, *i.e.* lassitude, and the sweats on the fall of the fever are sometimes large in amount; also during this period the body weight may decrease, or at all events, remain stationary. I found this in many cases where the fever increased, and in spite of the patient taking presumably a much more nutritious diet, still a slight weekly loss was observed. Some cases will react to the influence of the H.F. currents within twenty-four hours, others may take a few days. The more severe the case, the more quickly does the reaction take place. However much the temperature rises, it will generally be found down, or below the patient's usual normal, within forty-eight hours, so that the dose can be readily regulated and the patient only given as much as he can comfortably bear at a time. When the patient can be exposed to the currents for over half an hour daily for one week, and it is found that during the whole period the temperature remains

Digitized by Google



Digitized by Google

steady at normal and sub-normal, we may safely predict that the disease is, to say the least of it, arrested.

The chart shows a fairly typical temperature in a severe case; for the next three weeks it never rose over 99°; for the following eight weeks it never rose over normal, and generally was sub-normal; then daily observations were stopped. The weight increased 1 lb. during the first week; lost 1 lb. for the next two weeks, then steadily gained 1 to 2 lbs. for the next eight weeks. During the fourteenth week of treatment from the commencement of "high frequency" the patient "put on" $3\frac{1}{2}$ lbs. in that week. Her weight (in clothes) was 6 stone, and height 5 ft. 1 in. During the fourteen weeks' treatment she gained 1 stone $4\frac{1}{4}$ lbs. Two months after, with no treatment, she weighed (in clothes) 7 st. 13 lbs., which she has maintained for the past twelve months. The applications were fifty in number, and varied in dose from five to twenty minutes. After the third week twenty-minute doses were given on an average twice a week. In March, 1901, twelve months after, five applications of thirty minutes' duration, given on five consecutive days, could only raise the temperature to 99° in the evening, and it was 98·2° in the morning.

Forty cases were all treated in London, which can, at present, hardly be considered as a first-class health resort; but I am strongly of opinion that the application of high frequency electrical currents in sanatoria and like institutions will greatly swell the proportion of so-called cures. It is a remedy that should only be administered by medical men, as it requires as great care as any other therapeutic agent.

It is not possible to discuss in this short paper the scientific basis of electrical science. That knowledge can be obtained from the works of such men as Faraday, Clerk Maxwell, Elihu Thompson, Hertz and Tesla; and for the application to therapeutics from the writings of D'Arsonval, Begonié, Doumer, Oudin, Bordier, and others.

I purpose, therefore, to very briefly describe the apparatus I employ and the methods adopted to bring about the results I have placed before you.

The source of energy will, for most practitioners, be the electric light mains or secondary batteries—that is accumulators. One requires a large Rhumkorff coil giving a spark in air of 10 to 12 inches, a good motor interrupter, two Leyden jars, large and small solenoids and a D'Arsonval-Oudin resonator and suitable electrodes. The high frequency currents which we use are produced from the external armatures of the Leyden jars; the electrical energy of high pressure obtained from the induction coil is connected by the secondary terminals of the Rhumkorff coil to the inner coatings of the Leyden jars, which are so arranged as to allow a spark gap between them; the outer coatings of the jars being connected by means of a small solenoid, which in reality furnishes the currents of high frequency.

There are four ways by which the treatment may be carried out—by auto-conduction, auto-condensation, by means of local applications from the free end of the resonator of Oudin and the bi-polar method.

For auto-conduction is required a large solenoid of sufficient dimensions to envelop the patient, the two ends of which are connected to the outer

Digitized by Google

coatings of the Leyden jars. In this way a general electrification of the patient is brought about, and currents are induced in the body of the patient.

For auto-condensation the patient is placed upon a couch, having a large sheet of metal under the insulated cushions. This is made to form one armature of a condenser. The patient, when holding in his hands electrodes which are connected to the Leyden jars, forms the other armature of the condenser.

Resonator of mono-polar treatment. From free end of the resonator is obtained a brush discharge, or "effluve" similar to that obtained from the static machine—but of greater intensity and absolutely painless. This is directed by means of a brush electrode to the particular part of the patient requiring treatment. This brush discharge can be directed to the patient while on the auto-condensation couch, but should it be necessary to localise the effect of the "effluve" a suitable electrode connected to the apparatus must be used.

The fourth method, which I will call bi-polar treatment, is to use the flexible electrodes, which can be adjusted to any particular part of the body, and to use electrodes of such shape and size as the particular case demands.

The apparatus used at the demonstration was constructed by A. E. Dean, of Hatton Garden, London, E.C.

A REPORT ON THE TREATMENT OF TUBERCULOSIS BY DIRECT INTRODUCTION OF ANTISEPTIC MEDICAMENTS TO THE SEAT OF THE MALADY THROUGH THE PORES OF THE SKIN BY MEANS OF STATIC CURRENTS OF ELECTRICITY OF HIGH AND MEDIUM TENSION (CRÔTTE METHOD).

By J. Leffingwell Hatch, *B. Sc., M.D., formerly Assistant Demonstrator of Morbid Anatomy and Lecturer on Bacteriology in the University of Pennsylvania; Pathologist to the Philadelphia Hospital; late Sanitary Inspector for the port of Antwerp, Belgium; in the United States Marine Hospital Service, &c., &c.*

The application of static currents of electricity has for a long time been used in medicine, so also has formaldehyde; but just as it took a Columbus to show the world how to make an egg stand upon its end, so also it took a Crôtte to show us how to pass formaldehyde through the lungs by means of static currents of electricity of high and medium tension.

Ever since Koch discovered the tubercle bacillus in 1882, investigators all over the world have been trying to find a germicide that would be efficacious and at the same time innocuous to the patient.

Digitized by Google

Granting that such a medicament could be found, another difficulty arose, viz., the anatomy of the tubercle, which by its own growth throws out a wall of connective tissue around itself which is utterly impregnable to medicaments borne by the circulation.

Formaldehyde has been found to be a true germicide, but formaldehyde pure coagulates albumen, and consequently could not be passed directly into the blood current, as it would coagulate the blood and cause immediate death by forming thrombi.

Crôtte, after experimenting for a long time, has been able to make a preparation of formaldehyde which he is able to pass into the lungs through the tissues, in the form of a vapour, by means of static currents of electricity.

In order to prove that the formaldehyde actually reached the lungs and that the remarkable cures that he made were not due to electricity alone, he made experiments upon animals, and found the formaldehyde in the lungs after the animal had been subjected to the treatment.

In order to verify his own experiments he had other chemists present when he treated the animals, and allowed them also to test the tissues for the germicide.

I here submit a report from Mr. A. S. Wolf, medical chemist and toxicologist to various New York hospitals.

NEW YORK,
September 9th, 1899.

DR. FRANCISQUE CRÔTTE.

DEAR DOCTOR,—"I removed the lungs and organs of the guinea-pig you submitted to the formaline treatment, extracting same with distilled water, distilled the extract, treated the distillate with a drop of very dilute phenol and poured same over strong sulphuric acid. At the point of contact of the two fluids I observed a faint but positive pinkish coloration showing the presence of formaldehyde.

The organs of this animal showed a marked form of preservation, not the slightest evidence of putrefaction being evident."

(Signed) A. S. WOLF.

The above reference to the preserved condition of the specimen, after laying four days in a bottle without alcohol or other preservative, is another proof of the presence of formaldehyde, which prevents putrefaction.

NEW YORK,
September 9th, 1899.

DR. FRANCISQUE CRÔTTE.

DEAR DOCTOR,—"I received the lungs of the guinea-pig you treated with iodine; extracted same with distilled water and chlorine water, the same acidified and shaken with carbon disulphide; I obtained a marked violet red coloration, showing the presence of iodine"

(Signed) A. S. WOLF.

Digitized by Google

ANALYSIS OF THE VISCERA OF A GUINEA-PIG SUBMITTED TO THE CRÔTTE METHOD OF TRANSFUSION OF MEDICAMENTS.

In order to demonstrate the introduction into the organism of chemicals by means of the Crôtte method of transfusion by high tension static discharges, the following experiment was made :—

A guinea-pig was shaved on the surface of the abdomen and painted with a ten per cent. solution of bichloride of mercury ($HgCl_2$) and subjected immediately to proper electrical discharges for about one minute. The animal was killed after about two hours, and the lungs, salivary glands and bladder with its contents removed for examination as to their constituents.

I. *Lungs and Salivary Glands.*

The organs were reduced to a pulp with four times their weight of water and the liquid again separated by filtration. This submitted to the action of hydrogen sulphide (H_2S) yielded a black precipitate. This, upon separation and drying, proved to be insoluble in ammonium sulphydrate (NH_4HS), hydrochloric acid (HCl) nitric acid (NHO_3).

Partly soluble in boiling HNO_3 and entirely soluble in aqua regia, thus answering the tests to the presence of mercury in the filtrate.

II. *Bladder and Contents.*

The bladder was found to contain about one c.c. of urine of a rather dark colour. This was diluted with one c.c. of H_2O, filtered, and acidulated with one drop of HCl, and in it was immersed a piece of zinc with some gold foil wound around it in such a manner as to leave exposed a few strips of zinc. Twenty-four hours afterwards this was removed from the liquid, the gold foil unwound, washed, dried, and slowly heated in the closed end of a glass tube about 10 c. long. After a fraction of a minute a small amount of a whitish substance was seen to condense upon the upper and colder part of the tube; this, upon being examined with a magnifying glass, was found to be composed of minute globules of metallic mercury, thus proving the presence of this metal in the urine.

NEW YORK,
February 6th, 1901.

M. FRANCISQUE CRÔTTE.

DEAR SIR,—I have the honour to submit to you the results of the following experiments that were carried out in your laboratory.

Yours respectfully,
J. L. HATCH.

January 26th, 1901.

A guinea-pig was shaved on the surface of the abdomen and made fast to a board and laid on the insulated table of a static machine. Next, the

Digitized by Google

positive pole of the machine was connected by means of a chain with the back of the animal, and a sponge saturated with a solution of nascent iodine was fastened on the movable negative electrode. This sponge was brought in contact with the abdomen of the guinea-pig, held there for a few minutes, and then raised a few inches, next sparks were allowed to pass from the sponge to the body. The animal was then chloroformed and an autopsy immediately made.

The organs and intestines were tested with an acidulated starch solution and a characteristic blue reaction resulted, thus proving the presence of iodine, and also that it had been transfused through the tissues of the body by means of static currents of electricity of high tension.

J. L. HATCH, M.D.

January 24th, 1901.

Transfused a paper package and a thick muslin package, both containing a paste of *fécule de pommes de terre* with iodine, and on opening found the characteristic starch reaction.

I also performed the same experiment subsequently, substituting an acidulated starch paste for the *fécule de pommes de terre*, and obtained the characteristic blue colour reaction.

J. L. HATCH, M.D.

February 3rd, 1901.

A guinea-pig was shaved on the abdominal surface and fixed to a board as in the previous experiment, only in this case the head was covered with a thick cloth to keep the animal from respiring the substance to be transfused.

The connections with the machine having been established as before, a sponge saturated with 4 per cent. solution of formaldehyde was attached to the movable electrode. There were also large sponges saturated with the same solution placed in the hollow brass cylinders at the two different poles on the machine from which the chains lead to the electrodes.

The sponge on the end of the negative electrode was placed against the abdomen and chest of the animal and the current turned on. The transfusion was kept up for five minutes, after which time the animal was chloroformed and an autopsy immediately made.

The different organs were put in separate vessels and allowed to macerate in distilled water. I took a small portion of the lungs, crushed in the bottom of a test tube in a little distilled water with a glass rod, filtered the fluid, and tested the filtrate with Henner's test for formaldehyde.

I obtained a faint rose-violet ring, thus proving the presence of formaldehyde.

HENNER'S TEST.

To the suspected solution add the same quantity of a weak carbolic solution; pour this mixture upon concentrated sulphuric acid, and if the formaldehyde is present there will be formed at the point of junction of the two liquids a ring of rose-violet.

Digitized by Google

This test is very delicate—1 in 200,000.

I allowed the macerations to stand over night, and the next morning filtered and tested them seriatim. There were seven solutions in all; one from the lungs, one from the heart, and one from the stomach, one from the liver, one from the kidneys, one from the intestines, and one from the bladder.

In each and every instance I obtained the characteristic reaction; only the solution from the intestines gave the strongest reaction, while that of the heart gave the weakest.

This proves conclusively that formaldehyde can be transfused through the tissues—that is, transported to the internal organs by means of static currents of electricity of high tension.

The voltage that was used was about 2,000,000, with a very low amperage, a fraction of 1.

J. L. HATCH, M.D.

METHOD OF TRANSFUSION.

The antiseptic substances, for instance, formaldehyde, which are employed in the process-Crotte, are contained in suitable receptacles arranged in one of the electrodes connected with the electrostatic machine.

Accepted that this electrode is the negative pole of the apparatus, then clearly the antiseptic substances are located in the path of the current. In the process the negative electrode is placed at a convenient distance from the body of the person to be treated, and the electrode of opposite polarity is in a corresponding position on the opposite side of the body, so that the electrostatic currents of high tension pass from the accumulator of the machine through the receptacle containing the antiseptic, and from the electrode through the air between the electrode and the skin of the patient, then through the tissue and lungs at one side of the body, then through the tissues on the opposite side of the body to the skin, and then to the positive electrode, from which it returns to the machine, thereby establishing a closed electric circuit.

It is also a fact that continuous silent discharges of electrostatic currents of high tension, when passing through the air, are easily visible to the eye if the machine is operated at night in complete darkness, that static currents of high tension ozonise the air, and that this is also easily perceived, as the air, when ozonised, has a peculiar odour resembling somewhat the faint odour of sulphur.

It is well understood that the silent discharges of static currents of high tension readily pass through a stratum of air, such as that existing between the electrode and the skin of the patient, and that these discharges, on reaching the skin, pass readily through the tissues of the body, because the tissues and lungs contain conductive liquids.

As soon as these silent discharges enter the skin, then the currents have a better conductor over which to travel than the air between the electrode and the skin, and as the lungs constantly contain moisture these silent discharges

Digitized by Google

are well distributed and do not travel along a confined line, as when travelling along a metallic wire. The silent discharges upon reaching the skin on the opposite side of the body pass to the positive electrode, which is preferably made of metal, and therefore an excellent conductor.

It is entirely in accord with known phenomena that the antiseptics contained in the receptacle at the negative electrode are carried along by the electrostatic currents when the same are in the path of said currents. The porosity of metals to a fluid under direct mechanical pressure was demonstrated as early as 1661 by the Florentine Academy, by forcing water through metal sheets. In the Crôtte process, formaldehyde, which is a gas of peculiar pungent odour, is usually employed as the antiseptic. The antiseptic then being in the path of the current, and the static currents of high tension acting thereon, the conditions are analogous to those in the experiments of the Florentine Academy, with the difference that, in the present case a powerful flow of electricity at a high electrical pressure serves as a vehicle, or means for drawing along the antiseptic in molecular form through the metal, whereas, in the early experiment a direct mechanical pressure upon the fluid was employed. The current in this manner serves to convey the antiseptic through the metal of the electrode, and also through the thin stratum of air to the patient, and through the tissues to the seat of the disease, where it is absorbed; no perceptible quantity passes out of the body to the positive electrode.

The patient received this treatment every day, and it was remarkable to see the rapid improvement.

As soon as the activity of the bacilli is arrested the fever decreases, and the night sweats lessen, the appetite improves, and the tonic of the electric bath gives the patient renewed vigour.

Of course, special symptoms require other therapeutic measures in connection with the treatment, and each case has to be watched carefully, but the great principle of Crôtte's method is a simple one and is based upon electrical osmosis, that is to say, certain substances are carried along in the track of the electric current.

This principle is used in the Arts, and in Germany there is a large factory for making wood proof against the attacks of the marine teredo by driving a preservative into it by means of the electric current.

The Crôtte treatment appears to me to be the most successful of any that is known for tuberculosis. Of course it is not a worker of impossibilities, and cannot replace lost lung tissues any more than it can replace an amputated leg, but that it does arrest the specific inflammation is a patent fact.

So much for the theory of the treatment and the laboratory experiments which prove it. Now let me add the weighty evidence of the clinical facts.

I am only going to take the cases that have come under my observation during the past twelve months, and will not select special cases favourable to the treatment but take them all just as they come from the case book, seriatim.

Digitized by Google

Out of fifty-three cases I find the following results:—

20 perfect cures;
9 partial cures (left before treatment was complete);
11 disappeared altogether;
7 died;
2 refused;
4 under treatment at present and improving.

53

Subtracting the eleven that disappeared and the two refused, we have, out of forty patients treated—

50 per cent. entirely cured;
32·5 per cent. partially cured and improving;
17·5 per cent. mortality.

What a strong light is thus thrown upon the therapeutic possibilities of the future.

REPORT ON THE TREATMENT OF TUBERCULOSIS BY FRANCISQUE CRÔTTE'S METHOD.

By Dr. ALBERT SALIVAS.

Gentlemen,—At the International Congress of Medicine, held at Paris in August last, my honourable colleagues, Dr. Bertheau, of Paris, Dr. Ducamp, of Bordeaux, and Dr. F. T. Labadie, of New York, each read powerful papers in which all three affirmed with equal energy that the remedy for tuberculosis had at last been discovered. In support of their solemn assertions, they brought forward the clinical facts from which they had arrived at an unshakable conviction on this subject.

Dr. Bertheau set forth that for four years he had, assisted by Francisque Crôtte, followed the method of treatment of that inventor (as modest as distinguished) in hundreds and hundreds of the most serious cases, and that success had almost invariably crowned his efforts.

Dr. Ducamp in turn proclaimed that the method had yielded him in two successive years surprising cures in a number of cases regarded as absolutely hopeless.

These categorical statements and distinct proofs of my two fellow-countrymen were confirmed in a brilliant manner by Dr. F. T. Labadie, Delegate of the Medico-Legal Society of New York, who also had employed the Francisque Crôtte method for more than a year, and owed to it a number of really extraordinary cures.

Digitized by Google

Here we have three most serious practitioners, following their professions at great distances apart, and in entirely different regions, the two first on the old continent, the third in the new world, and whose testimony, strictly in accord, leaves no doubt upon the result obtained.

That is not all. The most significant fact is that the "Assistance Publique" of Paris has officially sent more than 2000 patients in the course of five years to Francisque Crôtte, who has gratuitously lavished his care upon them, just as he has done and is still doing in Germany and America.

Furthermore, during the past eight years, patients have been sent from all parts of the world to the inventor of this method by more than 200 physicians, whose unanimous letters of thanks and congratulations are a most conclusive proof of the value of the treatment. And now at the present moment two hospitals in Washington are adopting the method and the transfusing apparatus of Francisque Crôtte.

Are these not, gentlemen, notable facts impossible to pass over in silence, above all to-day, when the heart-rending question of tuberculosis is being exclusively discussed by this Congress?

Like my colleagues, Bertheau, Ducamp, and F. T. Labadie, I am enthusiastic over the method of treatment which has rendered them so many services. I have had the good fortune to be able to study it at length in the Clinical Hospital of Paris. I have been able to compare it with other methods, and have been able to judge it. Well, I say boldly, I declare with all the strength of my soul, with the conscience that I am fulfilling an imperative duty in so doing, this method is rapidly and surely triumphing over tuberculosis. All others, you know as well as I, are practically illusory; it alone cures. The numerous cases that I have seen treated, or that I have personally treated at the Clinical Hospital of Paris, have enabled me to recognise its marvellous efficacy, and once for all determined my ideas on that point. Since then, moreover, Dr. J. Leffingwell Hatch, of New York, Assistant Professor at the Pennsylvania University, and Dr. W. P. Geisse, in charge of the treatment of diseases in the hospitals of Bonn (Germany), have arrived at the same conclusions.

In what does the method of Francisque Crôtte consist?

This method (into the details of which it would require too long a time to enter here) was the subject of an interesting communication from the inventor to the Academy of Science of Paris in 1894, and is based on absolutely incontestable scientific grounds, as proved by the works of clever chemists and bacteriologists of America and Europe. It consists of a special process for the conveyance and transfusion of medicaments and metals through the pores of the skin at the root of the disease by static electricity at mean or high tension. Thanks to this, without danger to the patient, there can be passed into the system the most powerful antiseptics (therefore those which, as the formic aldehyde, are not able to be used *per os* or by the pores of the skin), and the metals indispensable to a good organic assimilation, such as iron and manganese.

The Francisque Crôtte method creates then quite a revolution in thera-

Digitized by Google

peutics, for it is efficacious not only for tuberculosis, but also for all other affections of microbic origin. Its inventor even obtained excellent results from it in three cases of cancer.

Our statistics indicate 100 per cent. of cures in the first degree, 75 per cent. in the second, and 30 per cent. in the third. Strengthened by what I have seen and what I know, and supported by loyal hearts, I am organising in Paris at the present moment a clinical hospital where those who have lost both health and fortune will be gratuitously attended to under the supervision of Francisque Crôtte.

At the present time there are also being fitted up model sanatoriums, where suralimentation will be undertaken and where the air in the sick wards will be filtered night and day, rendered aseptic by formic aldehyde vapours, and saturated with pure ozone. In these sanatoriums there is no need for high altitude; a healthy country and a good position are sufficient.

Under these circumstances, it would be almost criminal not to listen, to allow those attacked with tuberculosis to die from simple apathy, or disdainful indifference of a method which could save them, not to introduce that method into hospitals and sanatoriums.

I am here, then, gentlemen, in the name of the sacred interests of the unhappy victims of the scourge which you have nobly undertaken to combat, to ask you to form a commission charged with the propagation and popularisation of a method of which the past, short though it be, is already a most brilliant one.

REPORT ON SOME EXPERIENCES IN THE USE OF FRANCISQUE CRÔTTE'S METHOD IN THE TREATMENT OF CONSUMPTION.

DR. W. P. GEISSE (*Bonn, Germany*).

GENTLEMEN,—Incited by the Reports of Messieurs Bertheau, Ducamp and Labadie, on their success in the use of the method of Francisque Crôtte for the treatment of phthisis, communicated to the last International Medical Congress at Paris, I determined to put it to the test. I entered into communication with Monsieur Crôtte, and he was so good as to come from New York to Bonn in order to explain his method to me.

I have only treated a few patients, since I was not anxious to compile statistics, but wished rather to test the healing power of the method by careful observations made on a small number of patients.

Permit me to explain to you in a few words the essentials of the method. The treatment is electrical; the electricity used is generated by a strong influence machine, this electricity is, of course, statical. I refrain from giving you a detailed description of the machine: the one I use was constructed by van Houten and Tenbroeck, of New York, and modified by Crôtte.

Digitized by Google

How is it possible by means of electricity to influence the bacillus of tuberculosis and its result, consumption?

The success attained, as in all electro-therapeutical treatment, must in part be attributed to the suggestive influences. For is not a favourable psychical influence a factor in the curing of consumptives? Consequently the use of electricity, which can be seen and felt by the patient, is important as increasing the psychical influence which the personality of the doctor must necessarily have in the treatment of those suffering from tuberculosis.

Further, we must consider the purely physical effects of the electric current: it reinvigorates, it stimulates the nerves of the blood-vessels, which react to the stimulus by contraction and dilatation of their calibre; the muscles are caused to contract, and in this way an improvement in the circulation is effected, resulting in the improved nutrition of the different organs of the body.

Finally, those who place credence in the Crôtte method believe that the current possesses a specific healing property, by reason of its transporting influence and in the power of the sparks to kill the bacteria. Here we are in part dealing with hypotheses; we must allow the successes attained by this method to speak for themselves.

Still, these two facts are undoubtedly true: firstly, the electric spark exterminates the tubercular bacillus; secondly, the transporting influence of the statical electricity exists, if it is applied as Crôtte directs.

The medicament which is transmitted is formaldehyde in solution; the strong disinfecting properties of this substance are well known. When administered in carefully measured doses and precipitated according to the directions of Crôtte, formaldehyde loses its noxious properties without being deprived of its antiseptic properties. It is introduced into the diseased system by means of the electrical current, partly through the intact epidermis of the chest and back, and partly through the opened mouth; in addition, formaldehyde vapour and ozone are inhaled. In order to keep the body saturated with formaldehyde, the patient is in addition made to inhale carefully measured doses of the solution at home three times a day for a period of ten minutes; the usual form of apparatus for purposes of inhalation is used.

The treatment when the patient is sitting on the insulated chair in the consulting room is simple. We do not connect one pole with the earth, as is usually done, but we allow the whole quantity of electricity generated by the machine to pass through the body of the patient, either in the form of a spark, 15–20 cms. in length, to kill the bacillus, or in the form of a current for the transfusion of the antiseptic, the function of which is to render the toxins produced by the bacilli harmless.

The time at my disposal does not allow me to give an exact description of the treatment; as this is important, I will ask you to refer to the Reports communicated to the Congress at Paris last year.

I will only call your attention to one fact—namely, that the treatment is administered three times a week; the patient therefore is well under one's

Digitized by Google

control, and one can be certain that the orders are properly carried out, for it must be understood that in the treatment according to the Crôtte method, the hygienic-dietetic treatment is not neglected.

My experience in this method does not date back very far; it is only four months ago that I began my experiments; however, I have in this short time obtained very good results.

My patients belong entirely to the poorer class. Out of ten such patients, of whom eight are in the so-called second degree, and two in the so-called third degree of consumption with cavities, *two* are sure to be cured in a short time; two others have improved to such an extent that their cure is to be expected in a few weeks. They no longer cough or expectorate much. One of them, a woman who has had tuberculosis of the lungs for fourteen years, has recovered her strength to such an extent that she can manage her house, do the cooking, washing, sweeping, etc. Another patient, who six weeks ago discontinued the treatment in consequence of having to earn his living, showed marked signs of improvement.

Four other patients have more or less improved, and are going to be treated further. Only one patient, a woman, who discontinued the treatment nearly two months ago, showed no marked signs of improvement. She had consumption of the third degree, the galloping form with high fever, which could only be diminished temporarily; quinine and phenacetin were also useless. The patient, against our advice, left the hospital in order to go to relations in the country, and in consequence the treatment was necessarily discontinued.

I will not read long-worded reports to you, gentlemen, but will attempt to show the favourable influence of Crôtte's method on the leading symptoms of the disease.

The *nervous system* is favourably influenced to a surprising extent. Depression, hopelessness, imagined and real pains disappear at once, the sick body is endowed with new vitality, the eyes acquire a new brilliancy, the bearing is erect, the patient is certain to recover health. *The appetite* returns at once; sometimes after the treatment an irresistible hunger is felt, so that the patients cannot wait for their next meal. *Sleep* comes back, the *respiration* becomes easier and deeper, the *circulation* is improved, and the patients acquire a fresh colour; the *skin* reacts better and better to external impulses, the finger-nails lose their deadly white colour, and the natural colour returns.

The *fever* sinks after a few days' treatment, and does not return, provided the patient does not catch cold in between. This lowering of the temperature produced by the treatment is very striking. The patient whom I mentioned above as having galloping consumption, before the treatment had a body temperature of 102° F. to 104° F.; ten minutes later, after treatment with the electric current, her temperature was lowered by two, three, even four degrees; unfortunately, in her case the improvement only lasted for a few hours, but with the other patients this lowering of temperature was permanent.

Digitized by Google

The *feeling of strength* increases, the patients undertake daily longer walks and find it easier to go upstairs. The *irritation* causing the *cough* diminishes, *even* in the case of tuberculosis of the larynx; the *expectoration* diminishes in quantity after a slight temporary increase, due to the loosening effect of the spark and the increased activity of the lungs. Slowly but gradually the *number* of *bacilli* diminish; the *body* weight increases.

As far as the *physical symptoms* are concerned, what strikes one at once is that consolidations disappear, and in most cases the lungs are soon able to function quite freely. This condition may give rise to errors in auscultation owing to the fact that with the disappearance of the consolidations new rattles appear which were not heard before. Hand in hand with the *resumption* of the *breathing functions* by the lungs is the *easing of the action of the heart.* This is shown in a very striking manner by one of my patients, whose case I will just mention before bringing my Paper to a close. Both lungs were attacked from top to bottom. On the right side could be heard many rattles, while the apex of the left lung exhibited a cavity of the size of a hen's egg, with a well-defined Wintrich's change of sound; in the whole of the upper part of the left side could be heard many rattles, the consolidated lower part of the lung remained stationary at each breath, and the patient suffered intensely from want of breath, the expectoration was copious and very thick in consistency, the action impact of the heart was visible on a largely increased area of the chest, 100 or more beats took place in the minute.

Now after four months' treatment the right lung is almost free from rattles, the left lung has completely recovered its function, the change of sound over the lower part of the cavity no longer exists, the breathing associated with the presence of a cavity has lost its specific sound, the expectoration has diminished in quantity, and of the action of the heart only the impact of the apex is visible. The beats vary between 80 and 90 in the minute.

I hope after this progress to save the patient, provided no complications intervene. Such a complication has occurred since I have had him under my notice; on the right upper arm and forearm there appeared two swellings, which when opened with the knife were seen to be full of matter. These were removed in two sittings, one wound was sewn, and now only the scar can be seen; the other was kept open for a fortnight and healed without being sewn. The patient lost 1 kilogramme, but he soon regained his former weight.

Gentlemen, I am of the opinion that this short summary is sufficient to show you that there is something of real worth in Crôtte's method. You can see that it is not one of those treatments which almost daily appear only to disappear, but a genuine healing method, rich in prospects. As for myself, to be sure I have not completed my observations, but my mind is made up, and so will yours be when you have heard the favourable reports of my colleagues, Monsieur Salivas, from Paris, and Dr. Hatch, from New York, who have had many years' experience in Crôtte's treatment, and have by its agency saved many lives.

Digitized by Google

CONCLUSION.

The CHAIRMAN (Dr. C. THEODORE WILLIAMS): Ladies and Gentlemen,—In closing the Section I will say that I hope the work we have done has been of a profitable character. If you look at the range of the subjects we have dealt with and the variety of papers, I think you will agree we could not have done more. We began with climatology, the next day we had the treatment by tuberculin, in which Professor Koch, Dr. Heron, and others took part; yesterday we had a most admirable discussion, which was inaugurated, as you know, by Professor Clifford Allbutt, Dr. J. K. Fowler, and Dr. R. W. Philip. And I think the great feature has been not only the variety of the papers, but the practical application of them. The remarks of the speakers were of the most practical nature. To-day, of course, it has been different, because we have had a variety of papers, but even to-day there have been a number of speeches which will be of great use in the practical treatment of tuberculosis. So that I think we may look back on the past four days and say they have been well spent. We have had the privilege of the presence of intellects from almost every country in Europe and America, and the speakers have been able to condense their remarks so much that we have had a much larger amount of information and discussion than we could possibly have had if the papers had been long.

In conclusion, ladies and gentlemen, there is one duty which we all owe, and that is to the President. Our President was unfortunately not able to finish the business of the section to-day, but you all know, or most of you, that quite recently, just before the beginning of this Congress, he lost a dearly beloved son. When it was thought he was safe in South Africa, he became engaged in a skirmish in which he was killed. He was a brave fellow, and had gone forth to fight for his country, and his parents were beginning to look forward to his safe return covered with glory. Of course Sir Richard and Lady Douglas Powell were overwhelmed by the trouble, but Sir Richard is a man who would never desert his duty, and he has, notwithstanding his great loss, taken the greatest interest in this Section.

I will call upon Sir J. W. Moore to speak.

SIR JOHN WILLIAM MOORE, M.D.: Mr. Chairman, Ladies and Gentlemen,—After the words which have fallen from you, sir, a very few remarks will be sufficient to commend a resolution which I hold in my hand to your consideration. We have all admired Sir Richard Douglas Powell's dignity as president of this section, and we must consider ourselves fortunate to have sat under his sway. I think a great deal of the success which has attended the meetings of this section is due to his admirable courtesy, and, at the same time, firmness on all occasions. I was greatly struck by that on more than one occasion during the debates. Our hearts all go forth to him in his trouble and to the trouble which has fallen upon his family, and therefore it is that I have ventured to put these few words in writing and to read them to

Digitized by Google

you :—" Resolved, that the Section desires to express its high and grateful appreciation of the able, dignified, and courteous manner in which Sir Richard Douglas Powell has discharged his presidential duties under most trying and regrettable circumstances; and that the Section would convey to Sir Richard Douglas Powell and to Lady Douglas Powell an expression of its deep sympathy with them on the occasion of the death of their soldierly and heroic son in the South African campaign." Not a fortnight ago that young officer was in the enjoyment of full health, and doubtless looking forward to a happy union with his family. On Saturday week he fell fighting for his King and country.

Dr. C. Denison (Denver, Colorado): Mr. Chairman, Ladies and Gentlemen,—I think it is particularly appropriate and pleasurable for us to pass this resolution, and it gives me great pleasure to second it.

The Resolution was put and carried unanimously.

The Chairman: Ladies and Gentlemen,—That concludes our business, and I hope our next meeting will be at another congress on tuberculosis.

Digitized by Google

SECTION III.

PATHOLOGY, INCLUDING BACTERIOLOGY.

President.

PROFESSOR G. SIMS WOODHEAD, M.D., F.R.S.E.

Vice-Presidents.

Sir CHRISTOPHER JOHN NIXON, M.D., LL.D.
Professor J. G. ADAMI, M.D.
Professor D. J. HAMILTON, M.B., F.R.S.E.
Professor SHERIDAN DELÉPINE.
Dr. E. KLEIN, F.R.S.
Professor SIDNEY MARTIN, M.D., F.R.S.
Dr. J. W. SPRINGTHORPE.

Hon. Secretaries.

Dr. F. J. WETHERED, 83, Harley Street, W.
Dr. J. J. PERKINS, 41, Wimpole Street, W.
Professor RUBERT BOYCE, University College, Liverpool.
Dr. E. J. MCWEENEY, 84, St. Stephen's Green, Dublin.

TUESDAY, JULY 23rd, 1901.

PROFESSOR G. SIMS WOODHEAD, M.D., F.R.S.E.,

IN THE CHAIR.

ON THE RELATIONS OF THE TUBERCLE BACILLUS TO OTHER BACTERIA RESISTANT TO ACIDS AND TO ACTINOMYCES.

BY DR. MOELLER, *Belsig.*

DISCOVERIES that have been made in recent years of tubercle-producing bacilli resistant to acids and similar micro-organisms have made it impossible to diagnose the tubercle bacillus by the aid of the microscope alone. Before these discoveries were made, it had been customary to call every genuine bacillus that was resistant to acids and alcohol a tubercle bacillus.

It is true that what was known of the similar behaviour of the lepra bacillus, with respect to colour reaction, already showed that the tinctorial behaviour of the tubercle bacillus was to be regarded as dependent on its other biological qualities, that in course of time it might be possible to find other bacteria which were equally resistant to acids and alcohol, and with the same peculiarities with regard to colour reaction as the tubercle bacillus.

There has been a good deal of discussion as to the nature of this colour

Digitized by Google

reaction and its relation to the real substance of the bacteria. Koch himself says that, from a diagnostic point of view, the peculiar colour reaction of tubercle bacilli is at any rate of value, but that it is a great mistake to think that the etiological significance of tubercle bacilli stands and falls with its specific colour reaction.

Recently Klein, and later Marmorek, have shown that quite young tubercle bacilli are not resistant to acid and alcohol. Marmorek attributes this to the fact that young tubercle bacilli are not covered with that fatty and waxy envelope which, on the one hand, prevents the ordinary basic pigments from coming into contact with the protoplasm of the tubercle bacillus; and, on the other hand, prevents acids and alcohol from decolourising them when once stained. Klein thinks that the resistance to acids of the tubercle bacilli depends on the production of chemical substances on the part of the bodies of the bacilli. These, in the young tubercle bacilli, are wanting, and therefore they are susceptible to acids. Borrel, by experiments, succeeded in taking away the resistance to acids and alcohol of the tubercle bacilli. Under the protracted action of warm xylol a growth-like mass could be removed from the tubercle bacilli, which was resistant to acids and alcohol, whilst the tubercle bacilli thus treated had lost these qualities, though they were still capable of producing tubercle.

Whatever may cause this resistance to acids in the tubercle bacillus, it is quite justifiable to attribute the same tinctorial behaviour of the other bacteria, which are resistant to acids, to a like cause. But this mutual colour reaction is only the outer band that keeps the whole together; what is of much greater significance is, that there is more or less close resemblance among the bacteria, and with the tubercle bacillus, with respect to morphology and pathogenesis.

Bacteria resembling the tubercle bacillus, and known to us for years, are the lepra bacillus, smegma bacillus, and the organism of avian tuberculosis.

The bacillus of leprosy was first described, in 1877, by Armauer Hansen. It was found in leprous tubercles. It resembles the tubercle bacillus in form, but is generally of more uniform length, and shorter. It stains more readily than the tubercle bacillus, *e.g.*, it stains with aqueous fuchsin-solution, even at room temperature, which the tubercle bacillus will not do. Neisser, who has done much to make the bacillus of leprosy more generally known, gives Weigert's method of granule staining as the differential stain. Neither Hansen nor Neisser have succeeded in obtaining pure cultures. Bordoni-Uffreduzzi, in 1887, is alleged to have succeeded in cultivating from the marrow of the bones of a leper a micro-organism resembling the tubercle bacillus, even as regards resistance to acids. The cultures were not maintained. Cultivated bacteria from the organs of lepers, described rather later by Babes, and still more recently by Czaplewski, differ from the genuine bacillus of leprosy in their incomplete resistance to acids. Both authors maintain that the bacteria cultivated by them resemble the bacilli of diphtheria. In any case it is very doubtful whether these bacteria cultivated from leprosy are the genuine bacillus leprae.

Digitized by Google

The smegma bacillus was first discovered by Tavel and Alvarez, in 1885, in the normal preputial smegma, and also in the secretion of the outer skin, particularly where a collection of epithelium may occur, as in the anal and vulvar region, in the fold of the groin, between the toes, etc. The discovery of the bacillus was incidentally made in investigating an observation of Lustgartens, in 1887, on the syphilis bacillus. Morphologically and tinctorially the smegma bacillus closely resembles the syphilis bacillus of Lustgarten. This fact discounted the importance attached to Lustgarten's discovery, and subsequent investigations show that Lustgarten's bacillus has not been found in sufficient numbers, or with sufficient constancy, in the syphilitic tissue.

Smegma bacillus, according to Tavel and Alvarez, is morphologically exceedingly like the tubercle bacillus, can be stained by the same methods, but is less resistant to alcohol. Inoculation experiments on animals were without result, nor were the authors able to cultivate a pure culture. Laser and Czaplewski have cultivated (the former from the secretion of syphilitic affections, the latter out of gonorrhœal pus) micro-organisms resistant to acids, similar to diphtheria bacilli, which have been declared by both authors identical with the smegma bacillus. This statement has recently been objected to by C. Fränkel. Fränkel only calls those micro-organisms smegma bacillus which first attracted attention by their great resemblance to tubercle bacilli. This resemblance is wanting in Czaplewski and Laser's and also in his own cultures, which he described later. In form and other characters they are much more like a pseudo-diphtheritic bacillus. From my own personal observations I agree with Fränkel. I was not able to get a pathogenic effect in guinea-pigs either with the diphtheroid bacillus cultivated in pure culture from smegma or with cutaneous secretion containing the genuine smegma bacillus in abundance.

With respect to differential diagnosis, especially in reference to urogenital tuberculosis, the smegma bacillus is undoubtedly of greater importance.

As a micro-organism resistant to acids which has long been known, I may also mention the organism of avian tuberculosis. It differs from the organism of tuberculosis in mammals in the appearance of the cultures and the temperature conditions. Bacilli of mammalian tuberculosis will not thrive at 42° C., whilst bacilli of avian derivation will thrive at this and even higher temperatures. Fischel, who worked under Huppe's direction, says both micro-organisms are of one and the same kind as regards nutritive media. In consequence of the different physiological nutritive media of the colder mammalian bodies on the one hand, and the warmer avian bodies on the other, a distinction has arisen between the two kinds. By artificial cultivation Fischel succeeded in bringing about approximation in external qualities between the two bacteria; he succeeded in getting the organism of tuberculosis in mammals accustomed to a higher temperature, and in given nutritive media he got a resemblance in the appearance of the cultures, but as regards pathogenesis he could not transfer one to the other. Fischel says that he was able, with the organism of avian tuberculosis, to bring about a general tuberculosis in a guinea-pig, but the cultures started out of the organs of this animal were not

Digitized by Google

identical with those of avian tuberculosis. In my own similar experiments on animals I got the same results.

Tuberculosis in cold-blooded animals is in the same way to be regarded as a modification of the tuberculosis in mammals. Bataillon and Terre succeeded in cultivating tuberculosis in mammals and birds by means of passing it through frogs at room temperature. Lubarsch was able to modify tuberculosis in mammals by passage through frogs, so that cultures taken from the spleen of a frog grew at a temperature of 28–30°. Dubard also cultivated cultures taken from fish inoculated with mammalian tuberculosis which also thrived at room temperature. I was able to produce cultures from the spleen of a slow-worm inoculated with sputum containing tubercle bacilli, which flourished at 20°, but which ceased to grow at a temperature of 30° and over. The cultures resemble in appearance those of avian tuberculosis. I have not yet succeeded in obtaining cultures which got accustomed again to a temperature of 37°, even after passage through warm-blooded animals.

Recently it has been possible to demonstrate a series of micro-organisms in common articles of food, etc., which in many respects resemble the tubercle bacillus.

Tubercle bacilli were found in such appalling frequency in butter and milk that it led to an inquiry as to whether the genuine tubercle bacillus was really always found.

Petri and Rabinowitsch were the first to make such experiments. Both succeeded in getting a bacillus resistant to acids and alcohol from butter, that was *not* the tubercle bacillus. The micro-organisms described by these two authors are so materially alike that they are both generally described as the Petri-Rabinowitsch butter bacillus.

This bacillus has the appearance of an immobile bacillus in shape very like the tubercle bacillus, but which now and then is fatter. In the middle of the bacillus deeply coloured granules are sometimes visible as in the tubercle bacillus. Tinctorially the butter bacillus behaves in the decolourising solution exactly like the tubercle bacillus with the ordinary staining methods. In sections it is not so resistant to acids as the latter. The bacillus thrives at room temperature, and at incubation temperature there are visible signs of growth after twenty-seven hours. The pure culture of the butter bacillus differs distinctly on the usual fixed nutritive media from that of the tubercle bacillus; in bouillon and on Proskau's albumin free nutritive medium, on the other hand, the cultures of both bacilli are similar. If the butter bacilli are injected together with butter into guinea-pigs, sometimes changes occur in the bodies of the animals, which macroscopically as well as microscopically may be mistaken for genuine tuberculosis, in most cases peritonitis sets in with great formation of thickening. The bacilli injected in pure culture, generally speaking, bring about less important changes in guinea-pigs than those injected with butter. Langhans's giant cells, nests of epithelioid cells and typical tuberculous caseation are, according to Rabinowitsch, never to be found in the foci of disease. None of the animals injected with butter bacilli reacted to tuberculin. Another bacillus resistant to acids was isolated by Korn from

Digitized by Google

butter. According to the statement of the author, this bacillus, both morphologically and culturally, but especially in its action on animals, differs from the Petri-Rabinowitsch butter bacillus, so that it cannot be identified with it.

Latterly greater success has been attained in isolating bacteria resistant to acids from butter and milk, which, however, do not differ much from one another.

I have lately cultivated from milk in pure culture a micro-organism similar to the tubercle bacillus which does not differ materially from the grass bacillus II.

All bacteria resistant to acids cultivated from milk and its derivatives show a great resemblance to the grass bacillus. Taking into consideration the habitat of the latter, namely, cattle fodder, we are certainly justified in regarding the milk and butter bacilli as varieties of the grass bacillus. Such differences as there are, and these are very slight, may be explained by the passage through the bodies of animals.

I could prove the presence of the grass bacillus in grasses much used for cattle fodder and could cultivate them in pure cultures. As they were first discovered on timothy grass, phleum pratense, I have named them timothy bacilli.

The timothy bacillus has the shape of a slender little rod, sometimes slightly curved. Microscopically it is often impossible to distinguish it from the tubercle bacillus. Like the latter, it often contains deeply stained granules; oval unstained patches are found in its interior; it divides into branches, and sometimes has club-shaped swellings at one end. In the decolourising solution, as well as in sections with the ordinary staining methods, it behaves exactly like the tubercle bacillus. The timothy bacillus grows best at incubation temperature, but it thrives badly at room temperature. It thrives on all usual nutritive media. After thirty-six hours, at incubation temperature, patches of colonies are distinctly seen. The cultures generally vary considerably from those of the tubercle bacillus, but a similarity is sometimes attained if the timothy bacillus, after several passages through the bodies of animals, is cultivated at a uniform temperature of 37°. It then tends to become more like the tubercle bacillus in its slow growth. As regards pathogenesis, the timothy bacillus produces in guinea-pigs almost the same pathological changes as the Petri-Rabinowitsch butter bacillus. It has a different effect on rabbits. In them, if the timothy bacillus is injected into the veins and arteries of the animal, changes are produced which it is difficult to distinguish from true tuberculosis. The formation of giant cells, epithelioid cells, caseation produce a striking resemblance to true tuberculosis. Lubarsch says: "There can be no doubt whatever that it is quite impossible to distinguish with certainty by histological and micro-parasitic examination between timothy fungus tubercles and true tubercles, the distinction can only be brought out by culture. In all animals injected with the timothy bacillus a negative reaction to tuberculin was obtained. Another micro-organism resistant to acids, also found in fodder grass, is one

Digitized by Google

which I have called the grass bacillus II. It differs so essentially from the timothy bacillus that identity of the two cannot be admitted. Tinctorially, the grass bacillus II. behaves like the timothy bacillus or the tubercle bacillus. In older cultures it loses somewhat its power of resistance to acids. Morphologically it takes the form of small rods, similar to those of the tubercle bacillus, but sometimes rather thicker. One peculiarity is its tendency to grow out in long rodlets and branches, a tendency often met with in the older cultures of the tubercle bacillus. In appearance the cultures of the grass bacillus II. bear a strong resemblance to the Petri-Rabinowitsch butter bacillus. It grows most quickly at incubation temperature, but also thrives at room-temperature. In guinea-pigs it produces the same changes as the butter bacillus; it shows the greatest virulence in milk cultures. Experiments made by Freymuth have shown that the injection of the grass bacillus II. into cold-blooded animals—fishes, frogs, lizards—will cause tuberculous disease. The bacilli found in the bodies of animals have assumed a more slender shape, so that morphologically they cannot be distinguished from the tubercle bacillus.

I should like also to point out, as a variety of the grass bacillus, micro-organisms resistant to acids found in the excreta of animals, and therefore called the manure bacillus. I found it in a manure heap which had lain for months, and also in the fresh excrementa of cows, donkeys, and other herbivora. The manure bacillus bears a morphological and tinctorial resemblance to the timothy bacillus; in growth of cultures and in pathogenesis it also resembles the grass bacillus II.

In this *résumé* I prefer to pass over the lepra and smegma bacillus on account of their relatively rare occurrence, and because so little is known of their cultural growth, and the avian and cold-blooded mammalian tubercle bacillus on account of their unimportance as regards differential diagnosis.

We are, therefore, left with grass bacilli as primitive form, with its varieties, milk and butter bacilli and manure bacillus. With respect to colour reaction they all, with slight deviations, behave like the tubercle bacillus. The same may be said of their morphology. Though in form the different species vary more or less, it is often impossible microscopically, under certain conditions, to distinguish them from the tubercle bacillus, and the tubercle bacillus itself varies according to the nutritive medium that it finds. I have made innumerable examinations of sputum in which I have seen the rods somewhat shorter, sometimes longer, sometimes thicker, and sometimes thinner. B. Fränkel first drew attention to the fact that the tubercle bacillus in the florid form of tuberculosis generally took the form of short rods. Those forms which deviate from the ordinary bacillus shape, such as rodlets, branching forms, keel-shaped swellings, as well as oval patches in the middle of the bacilli which will not stain, deeper-dyed granules, etc., are met with in the tubercle bacillus, as well as in other bacteria resistant to acids.

But in cultural growth they differ materially; in the first place, in the appearance of the cultures; even when now and then a more or less close resemblance has been attained through the methods of cultivation, the

Digitized by Google

tuberculosis culture shows such uniform characteristics that it is easy to distinguish it from all the others. Then it stands apart in its subtle behaviour with regard to temperature. Whilst other bacteria resistant to acids thrive at room temperature, the tubercle bacillus requires incubation temperature. The position of the tubercle bacillus is unique with respect to its excessively slow growth. Other bacteria resistant to acids form visible colonies at the latest after twenty-four hours at incubation temperature, whilst the earliest moment that growth has been observed in the tubercle bacillus is only after several days, even under the most favourable conditions of frequent transplantation on specially chosen nutritive media. This exceptional behaviour of the tubercle bacillus is very valuable from a differential diagnosis point of view. Several authors say that sometimes bacilli resistant to acids were found in the sputum in morbid conditions of the respiratory organs, e.g., in gangrene of the lung, which, on closer examination, did not prove to be genuine tubercle bacilli. Fränkel and Pappenheim proved this by dissection; Rabinowitsch proved conclusively, by pure culture of the bacteria, that it was probably a variety of their butter bacillus.

I have frequently found these pseudo-tubercle bacilli not only in sputum from the lungs, but also in mucus from the nose and pharynx, coating of the tongue, sordes on the teeth, secretion on the tonsils.

In those cases in which physical symptoms are entirely absent, and the suspicion of tuberculosis is based on the existence of bacilli resistant to acids in the secretion of the respiratory organs, the diagnosis may be confirmed by the following simple method, based on the slow growth of the tubercle bacillus and its peculiar temperature requirements. The secretion in question is mixed with nutritive bouillon, and kept at about 30°. If there is a visible increase in the bacteria resistant to acids, it is certain that it is not the genuine tubercle bacillus. Sometimes when the sputum is mixed with certain nutritive media, the tubercle bacillus grows at incubation temperature. This proliferation, due in all probability to the importation of globulin-like substances from the body, is, however, exceedingly small, and ceases altogether after, at the latest, forty-eight hours; whilst in the pseudo tubercle bacilli a persistent further proliferation takes place at 30°. There was no pathogenic effect noticeable either in the bacteria isolated by Rabinowitsch in gangrene of the lung or in those found by me in mucus from tonsils, nose, and pharynx.

Different occurrences in ordinary life bring out the differentiation of pseudo-tubercle bacilli from genuine tubercle bacilli even more than at the bedside. I should like to give two examples.

For years our most important articles of food—milk and butter—were looked upon with lively horror, because all the bacteria resistant to acids they contained were supposed to be tubercle bacilli.

The isolation of the manure bacillus showed the untenableness of the conclusion that the bacteria resistant to acids found in the excrementa of cattle were derived from the bovine tuberculosis existing in the animals concerned.

As regards pathogenesis, all bacteria resistant to acids have this in common,

Digitized by Google

that they cause a tuberculous disease in the usual animals experimented on, the genuine tubercle bacillus always, but the pseudo tubercle bacillus only in a limited number of cases and under certain conditions. The whole appearance of pseudo tuberculosis in the bodies of animals frequently bears macroscopically a striking resemblance to genuine tuberculosis. But in the tubercles themselves there is a visible difference; whilst the genuine tubercles are of a solid proliferating kind, the pseudo tubercles are of a more inflammatory character with tendency to abscess formation. The typically histological condition of genuine tuberculosis giant-cells, epithelioid cell nests, caseation is only met with in pseudo tuberculosis in very rare cases, when, under given conditions, the bacilli are introduced into the bodies of animals.

Another peculiarity of the tubercle bacillus I should still like to refer to. If, together with butter, it is injected intraperitoneally into animals, typical tuberculosis does not result, but peritonitis sets in accompanied by extensive formation of induration, exactly the same as follows the injection of pseudo tubercle bacilli and butter.

Though the obvious resemblances between bacteria resistant to acids and the tubercle bacillus are so many and so great, they only produce an apparent relationship. As far as the real, I might say, characteristic of the tubercle bacillus is concerned, its pathological effect on the human organism, its etiological significance in tuberculosis, it occupies a perfectly unique position among bacteria resistant to acids.

Though the occurrence of pseudo tubercle bacilli in the human organism, also in diseased organs (gangrene of the lungs, disease of the tonsil), has been repeatedly proved, no etiological significance has ever been attached to them.

By artificial cultivation tubercle bacilli and timothy bacilli, *e.g.*, may be made to resemble one another so much in their external qualities that the one acquires the qualifications of the other. The tubercle bacillus may become accustomed to a more rapid growth at a lower temperature, and the timothy bacillus may acquire the slow growth of the tubercle bacillus, and in appearance the cultures may be alike, and yet they really remain two separate kinds. They cannot be transferred, the one to the other. However perfect our technique may be, nature after all is the best cultivator, who will not be controlled, and what nature has willed to separate, it will be difficult for us to unite.

The tubercle bacillus has another peculiarity, and that is its method of propagation. A saprophytic growth is only demonstrable in cultures. It can only be found in the products of disease, or where contamination has occurred from them; whilst most of the bacteria resistant to acids noticed in this summary show a preponderating saprophytic growth.

The pleomorphism of the tubercle bacillus has long been proved. Soon after tubercle bacilli were known to exist at all, forms of growth were noticed by more than one author which deviated from the ordinary rod shape, though staining by the same method. Rod-shaped, branching and club-shaped swellings were noticed in pure cultures prepared from sputum from phthisical patients, and from fragments of tubercular lung cavity when treated with decolourising agents. Recently it has been possible, by infection with tubercle

Digitized by Google

bacilli, to produce formations in the tissues of animals, by means of which a close relationship between tubercle bacillus and actinomyces could be proved botanically. Babes gives an account of the actinomycotic conditions found on dissecting the tissues of rabbits after subdural inoculation of tubercle bacilli. Friedrich arrived at the same results by intra-arterial injections. Lubarsch and Schultze, after making tests, confirmed these statements, and it was also discovered that actinomycotic formations appeared with local injections into the different organs. More or less plainly developed actinomycotic formations were observed, according to the degree of virulence of the inoculation material used, the modes of injection, and the time the animal was experimented on. All authors testify to the great resemblance of this condition with actinomyces bovis. There is one difference, however, according to Friedrich and Nöske, and that is, that with tubercle actinomyces it is only in very exceptional cases that a direct relation of a bacillus to one of the swellings can be proved, whilst, with actinomyces bovis, the proof of the connection between club-formation and mycelium is comparatively easily and more frequently attained.

The tinctorial behaviour of these actinomycotic forms of the tubercle bacillus is variable; the club formation only in very rare cases shows itself resistant to acids. Staining by Ziehl-Neelsen solution does not always succeed in demonstrating the club formation, while the Gram-Weigert, Friedrich, and Birch-Hirschfeld methods yield good, reliable results in dealing with actinomyces.

Lubarsch, by exhaustive experiments, has established the fact that in the case of other acid-fast bacteria, fibres, branched forms and clubs have always been observed when pure cultures have been treated with decolourising reagents, and actinomycotic formations may likewise be produced, provided they are injected into animals in the same way as the tubercle bacillus—a proof that the pseudo-tubercle bacillus bears also a close connection to the actinomyces, whereby again a relationship is set up between the pseudo-tubercle bacillus and the tubercle bacillus.

Although we have succeeded by artificial means in producing in the tubercle bacillus the special forms of growth whereby it is proved to be one of the more highly organised myces, we have not yet succeeded in keeping it *constant* in this form. Perhaps in course of time the special conditions required will be discovered. In the meantime the tubercle bacillus is of the greatest importance in the stage in which we know it best, namely, as a parasite in the form of rodlets.

Digitized by Google

THE MORPHOLOGICAL AND PHYSIOLOGICAL VARIATIONS OF THE BACILLUS TUBERCULOSIS AND ITS RELATIONS—

a. To other Acid-fast Bacilli.
b. To the Ray Fungus and other Streptothriceae.

By WILLIAM BULLOCH, M.D., *Bacteriologist to the London Hospital.*

It is certainly the immortal service of Robert Koch to have shown in his paper on the "Ætiology of Tuberculosis," one of the most complete researches ever published in medical science, that the only immediate cause of tuberculosis is the tubercle bacillus, and to-day this is everywhere accepted as a fundamental fact in pathology. The complete study of this micro-organism from as wide a standpoint as possible must form the foundation-stone of the work of this Congress, as it is upon a thorough and extensive study of the biology of the parasite that the prophylaxis and rational therapeutics of tuberculosis must take its stand.

In no small a degree, then, the pivoting point of the work which the Congress has attempted lies in this Section, and in the sub-divisions of this Section, on the knowledge of the actual ætiological agent—Koch's bacillus.

The responsibility which has fallen on the hands of Dr. Moeller and myself is therefore great, and for my own part I regret that an English bacteriologist of greater standing and experience has not undertaken the task, especially as the knowledge of the tubercle bacillus is at the present day ramifying in so many different directions, and these latter-day currents of thought are to-day so difficult to gauge and interpret correctly.

Originally Koch described his bacilli as "invariably appearing in the form of small rods of the length of one-quarter to one-half the diameter of a red blood corpuscle (1·5–4 μ); although their length varies, their breadth is pretty constant, provided that the same method of staining is used. The tubercle bacilli are not, as a rule, quite straight rods: they usually show slight bends or breaks, and often a gentle curve, which may increase in the longest forms to such an extent as to reach the first stage of corkscrew structure." Koch also describes spores in the tubercle bacilli in the form of oval bodies bounded by a fine coloured line, from two to six being present in a single bacillus.

Now, later researches have shown that the structure of the tubercle bacillus is not so simple. It has, in fact, been doubted, and apparently on good grounds, whether the tubercle organism is a bacillus in the strict sense of the word, or whether it belongs to a more highly constituted division of fungi; and in recent years quite a number of bacteriologists have placed it, from a taxonomic point of view, in or near the streptothriceae or actino-mycetes (Lachner, Sandoval), and they now prefer to speak of it as the

Digitized by Google

tubercle fungus. Of the several new names given to it, perhaps that which is most widely used is the Mykobacterium Tuberculosis (Lehmann and Neumann).

Before, however, accepting this rashly, it is well to study the grounds on which this important departure from the original standpoint is based. To a certain extent this is due to the observations of Babes (1882), Nocard and Roux (1887), Metschnikoff, Klein, Czaplewski, and others, on the existence of branched forms and filaments with clubbed ends.

In particular, Metchnikoff found that the so-called bacillus elongated into short filaments, and this not only in sputum, but also in the splenic pulp of tubercular birds. While admitting the difficulty of correctly interpreting these forms, Metschnikoff thought that the evidence of their being degenerative was not sufficient, and that the tubercle bacillus is not an end stage, but only a condition in the developmental cycle of a filamentous fungus which he proposed to call Sclerothrix Kochii.

Similar observations were likewise made by Fischel and by Bruns, both in the case of the bacilli in the tissues and in cultures, and the latter of these observers expressed the view that such aberrant forms really belonged to the saprophytic vegetation of an organism which, as a parasite, appeared in the form of rods. A further stage in the development of this doctrine is represented by the researches of Coppen-Jones (1895), who concluded :—

1. That in the tissues and secretions the organism occurs chiefly as a short or long rod, which reproduces by fission.

2. Occasionally in the sputum, and always in old cultures, appear filamentous forms which are not septate, but show true dichotomous branching.

3. These filaments occur only on the surface of the medium, whereas in the depth there are only rods.

4. The rods do not contain genuine endospores, but they, as well as the filaments, contain bodies which possess many of the physical qualities of spores.

Researches subsequent to these have only led to a more general acceptation of these ideas, which have been placed on a still sounder basis by the work of Babes and Levaditi, Friedrich, Schultze, Lubarsch, Moeller, Ledoux-Lebard, and others. For example, Babes and Levaditi found these aberrant forms in rabbits injected with cultures under the *dura mater*. For from one to three weeks the organisms were found mostly in rod forms, whereas by the end of the fourth week a marked change had taken place in so far that the parasite now presented a marked radiated appearance, the elongated rods being branched and their free ends clubbed. By high magnifications the clubs could be followed deep into the bacillary focus.

Friedrich and Nösske introduced cultures of several races of tubercle bacilli into the left ventricle of the heart of rabbits, and observed a very pronounced development of these remarkable clubbed actinomycotic forms. As a difference from other observers, they found that the best radiated fungi were developed from the injection of the youngest and most virulent cultures, so that they were unable to look upon these actinomycotic forms as

Digitized by Google

being retrograde in character. They thought, rather, that such morphological alteration of the tubercle bacillus was an expression of an active conflict between the tissues and the parasite, and it can best be seen with virulent cultures, rapid dissemination in the blood stream and settlement in the viscera without a prolonged latent period in the tissues. After tuberculisation of the host had occurred actinomycotic forms of the parasite disappeared.

On repeating these experiments of Babes and Levaditi and of Friedrich, Schultze was led to the conclusion that virulent cultures do lead to the development of the so-called actinomycotic forms, or, at any rate, that the occurrence of such forms is not a sign of attenuation of the virulence of the parasite, and the same facts were made out by Lubarsch for several modified or allied forms of micro-organisms. So that there can no longer be much doubt on this point. And apparently upon morphological grounds alone it seems justifiable to classify the tubercle parasite among the group of higher fungi known as actinomycetes. And this is all the more probable as in its anatomical effects the tubercle bacillus is allied to several species of Actinomycetes, one of the chief characteristics of the pathogenic members of this group of fungi being the production of specific productive inflammatory new growths of a granulomatous type.

Granting, then, that the tubercle bacillus is really a member of a higher type of fungi, the interpretation which is put upon the clubs is varied, as it is and has been for the true ray fungus. As is well known, the view which has dominated this subject for a number of years in the case of the actinomyces fungus is that of Bostroem, that the clubs result essentially from regressive change in the sheath of the organism, and this view is shared in the case of tubercle by Alfred Fischer and others. The extreme opposing view, that the actinomycotic arrangement is the expression of the highest development of the fungus, has been held chiefly by Friedrich, while Lubarsch and Schultze take an intermediate stage in looking upon these actinomycotic forms as having the significance of a malformation from interrupted growth.

However seductive the theory may be that the tubercle bacillus, so called, is really a filamentous fungus with a relatively complicated structure, it is perhaps wiser to wait for more conclusive evidence, as the issues at stake are so vital for the doctrine of the origin and transmission of tuberculosis, and the work of this section of the Congress will have been crowned with success if unquestionable evidence of this view is established.

The possibilities of obtaining a further support for this theory may be looked for in the behaviour of the organism outside the body in the culture tube. Originally Koch recommended, as the result of failures with other media, the use of stiffened blood serum as the most suitable medium for the isolation and continuance of cultures of the bacillus. Later, Nocard and Roux showed that the addition of glycerine to various media, but especially to agar, materially enhances their nutritive value, and, since their publication, glycerine agar and glycerine bouillon have been extensively used for continuing, at any rate, the subcultures once obtained on Koch's medium. In

Digitized by Google

any case the isolation of fresh strains of tubercle bacillus is not easy, nor is it always possible in a given case. Apparently the main difficulties depend on the slow growth of the organism, so that if other and more rapidly growing bacteria are present—and they frequently are—they may overgrow the medium and render it useless before the tubercle bacillus has begun its first divisions. Even if the tubercle bacillus is unassociated, the slow growth may be entirely interrupted by the drying up of the medium, although this can be overcome by the closure of the tubes. Even this precaution is not altogether for the welfare of the bacillus, as, being aerobic, a free supply of oxygen is prevented, and it may just turn the balance and inhibit the growth.

By cultivating the organism in glycerinated potato in sealed tubes, Pawlowsky (1888) showed that in general good growths could be obtained, and this method has since been extensively used and has generally yielded most satisfactory results. Occasionally, however, the organism refuses to grow, even if the medium is freely oxygenated as recommended by Sander, Obici, and others; and in some cases of this class the failures seem to depend on the quality of the potato used, partly also on the reaction. In other cases the want of success seems to be due to the particular race of the bacillus used. As substitutes for potatoes, glycerinised potato-juice agar or bouillon have been recommended by Lubinski (1896), although, as Tomasczewski (1899), in a very careful research, has pointed out, good cultures are the exception rather than the rule. Similar conclusions have been reached by Ficker, who found, however, that glycerinised potato serum was especially favourable. In a comparative study of media Ficker used solid or fluid media containing sputum, and in general found that the luxuriance of the cultures was largely dependent on a certain definite degree of acidity in the medium, and on media composed of cerebral substance or containing certain proportions of it excellent cultures were obtained.

In recent times Hesse (1899) has strongly recommended, especially for isolating the bacillus from sputum, a medium containing a soluble albumin preparation known in commerce as "Nährstoff Heyden." Upon agar containing 0·5 per cent. of this substance along with 3 per cent. of glycerine, he obtained direct from tubercular sputum characteristic microscopic growths in one to three days. Even where there are other bacteria the commencement of the tubercle growths can be demonstrated in contact preparations in five or six hours. Now, although this result has been confirmed by Bronstein, Jochmann, Carl Fränkel, and others, it does not appear that Hesse's medium is an optimal one for tubercle bacillus, at least, not as a substratum for subculturing the organism. In this it is unquestionably inferior to glycerinated serum, although for cultivation from sputum it is distinctly better. In the place of Nährstoff Heyden other substances of a somewhat similar nature, if differing in origin, have been used with more or less success by Fränkel. Media containing somatose, eucasin, nutrose, sanatogen, plasmon, mutase, and used for subculturing tubercle bacillus, were mostly inferior to Heyden's Nährstoff, and certainly so to glycerine serum.

For isolating tubercle bacilli from sputum, Fränkel found that Hesse's

Digitized by Google

medium or plasmon agar occupied the first place, glycerine serum being inferior. From the researches published during the last two years there can be little doubt that on Hesse's medium, under certain special conditions, cultures can be obtained with a rapidity not possible by older media.

The subject of the simplest food-demands of the tubercle bacillus has also been studied, especially by Kühne and by Proskauer and Beck (1894), who found that luxuriant growths may be obtained in proteid-free media. In particular, the two latter observers have shown that the carbon and nitrogen demands of the bacillus may be well fulfilled on a medium consisting of magnesium citrate, ammonium sulphate, glycerine, and mannite, or even in a fluid containing only—

Commercial ammonium carbonate . .	0·35 per cent.
Primary potassium phosphate, KH PO .	0·15 „
Magnesium sulphate	0·25 „
Glycerine	1·5 „

Although at first the organism did not take to its altered surroundings, it later on grew luxuriantly, showing to what an extent it is possible to acclimatise an organism even like tubercle bacillus. In these experiments, which were apparently made with subcultures only, the elective affinity of the bacillus for glycerine was shown in so far that it was impossible to replace this substance even by bodies allied to it chemically.

As a rule the bacillus grows best about 37° C., the minimal and maximum limit being 29° and 42° respectively. This relatively high minimal temperature is against the supposition that the bacillus multiplies extra-corporeally, at least in temperate climates. It may be possible to lower the minimal temperature artificially and by degrees, but, ordinarily speaking, as it leaves the body of man and animals its possibility of growth is limited to a relatively small and at the same time a relatively high range of temperature.

In regard to the tinctorial relations of tubercle bacillus, it was very early found that the ordinary watery or hydro-alcoholic solutions so extensively used for the demonstration of other bacteria penetrated into the tubercle bacillus imperfectly and with extreme slowness. Even the addition of alkali to the stain as recommended by Koch possesses only historic importance.

It is the great service of Ehrlich to have shown in 1882 that the tubercle bacillus possesses peculiar properties as regards staining; of these the most remarkable is unquestionably the ability of the stained bacillus to resist the decolourising action of acids, even when the latter are applied in heroic doses, such as 33 per cent. nitric acid, or 25 per cent. sulphuric acid. The resistance to acids is, however, not limited to mineral acids, but is also exerted towards organic acids—acetic, formic, oxalic, tartaric, lactic and picric. This resistance to acids, or "acid-fastness" (*Säurefestigkeit*), has been the subject of much investigation, and it is entirely upon this principle that the microscopic diagnosis of tubercle bacillus is made.

For the actual stain fuchsin has been chiefly used in combination with anilin water (Ehrlich) or with carbolic acid, although other bodies may also

Digitized by Google

be used, such as paratoluidin, metatoluidin, benzaldehyde, resorcin. In all cases the penetrating properties of the stain are much increased, so that once stained the organism is not decolourised by acids and is thus "acid-fast."

From the fact that the stained bacillus is acid-fast Ehrlich has formulated the idea that this property is essentially produced by some substance surrounding the actual capsule, and whereas this hypothetical covering is permeable for anilin water, alkalies, etc., it is impermeable for acids. Hence the bacilli retain the stain while the surroundings are decolourised. By counter-staining or after-staining with a different colour a marked contrast between the tubercle bacilli and other micro-organisms can be obtained depending entirely on this acid-fastness.

At first a certain amount of doubt existed whether pure watery solutions could stain the tubercle bacillus at all, but later it was found that, if used hot, such solutions do stain, but the colour is only to a slight extent fast, especially towards strong solutions of sodium sulphite. In such cases the subsequent treatment by sulphite will be found to lead to decolourisation within one and a half hours, whereas if anilin water has been used in the composition of the stain, soaking for twenty-four hours in sodium sulphite is borne without effect. If the preparation is immersed in the sulphite for longer periods decolourisation ensues slowly and not uniformly, in so far that parts of the bacillus, of oval form and situated mostly at the pole, retain the stain, and in this way are sharply demarcated. These egg-like bodies may, in fact, retain the stain under the influence of sodium sulphite for eight or ten days (Ehrlich).

The exact changes which go on when the fuchsin-stained bacilli are immersed in mineral acids is evident from test-tube experiments. When sulphuric acid, for example, is allowed to act on the mono-acid rosanilin salts they are converted into the soluble brownish-coloured tri-acid compounds. In the case of tubercle bacilli this change takes an appreciably longer time than in the case of other bacteria, and apparently it does not set in till some change in the permeability of the bacillary membrane has been induced by the acid. The washing of the decolourised bacilli in water leads to a decomposition of the tri-acid compound with re-formation of mono-acid fuchsin, and the bacillus again appears red, as the fuschin does not diffuse out of it. There appears, then, some good reason for the assumption that there is some peculiarity in the limiting membrane of the tubercle bacillus, and that there is some substance in the bacillus which gives to it this peculiar acid-fast property. Now, so far as is known, this acid-fastness is exhibited on all media, even the most simple, but the degree of the fastness is variable, depending on age, race, and possibly on other factors; and it has also its time limits.

At first this *Säurefestigkeit* was looked upon as a specific character of Koch's bacillus, but in a short time other acid-fast bacilli were discovered, and, in fact, tissues other than bacilli, *e.g.*, the outer layers of the epidermis, certain hairs, the capsule of the coccidium oviforme, ova of tape worms, etc.

That the tubercle bacillus should alone among bacteria be acid-fast would

Digitized by Google

indeed be strange, and Koch in 1884 stated that it was "not improbable that in time other bacteria may be discovered which have the same staining properties as the tubercle bacillus"; and already a large number of such organisms are known, to mention only bacillus leprae, bacillus smegmatis, the butter fungi found by Petri and Rabinowitsch, and the acid-fast organisms found in grasses and manure by Moeller. Similar acid-fast non-tubercular bacilli have also been found in lung gangrene by Rabinowitsch, in certain chronic eye diseases by Ginsberg, in the nasal cavities by Karlinsky, and it is not unlikely that the number of these will be still further increased. From the different sites in which such organisms have been found their distribution in nature appears to be wide. And not only may they resemble the bacillus of Koch in form and in fastness to acids, but some of these pseudo-forms may induce nodular diseases like tubercle in animals, and in the lesions in animals the fungus may be found in actinomycotic form, like the ray fungus or true tubercle bacillus. The existence of such double forms and their wide distribution serve to make one cautious in giving a positive diagnosis of tubercle bacillus from microscopic examination only. For human tubercle, especially phthisis, the existence of these pseudo-forms probably does not affect the microscopic methods of diagnosis, but in the diagnosis of supposed tubercle bacilli in objects which from their nature and source are liable to be contaminated with tubercle-like organisms, recent investigations have shown that great care must be exercised. Even the increase in the severity of the microscopic test by the application of alcohol after the mineral acid is not to be relied upon as a differential means between the tubercle bacillus and its "doubles." Non-tubercular acid-fast bacilli may be as acid-fast as the most fast of the genuine T. B. In all cases where there is a possibility of doubt it is imperative to make cultures, and proceed to the application of the experimental inoculation test, with subsequent recultivation from the animal body.

Since this *Säurefestigkeit* is for the organisms which possess it a highly characteristic feature, it becomes necessary to study the causes to which it is due, as in this way a closer analogy between acid-fast bacilli may be established.

In general, following Unna and Klebs, it has been supposed that the acid-fastness depends on the presence in the bacillus of fat, and there can be no doubt that fatty acids, at any rate, are important constituents of the bacilli, as has been shown especially by de Schwenitz and Dorset, Rüppel, Koch, Aronson and others. The application of fat stains (Sudan III.) has in fact been recently recommended for staining the tubercle bacillus (Dorset, Sata). Aronson (1898) has, however, shown that the body which gives the acid-fast character to the tubercle bacillus is of the nature of a wax. If the bacilli are treated with alcohol-ether mixture alone they preserve their acid-fast characters, whereas if the mixture is allowed to act in the presence of hydrochloric acid, they are no longer acid-fast.

These experiments bring us to the deeper and more difficult problem of the exact chemistry of the tubercle bacillus. Hitherto many of the researches

Digitized by Google

on this subject have suffered from imperfections of technique and from the difficulties naturally inherent in the subject, so that a revision of some of the older work would seem urgent, especially as it is now apparent that the chief deleterious substances in the tubercle bacillus are endobacillary. Of the newer researches perhaps the most important are those of Rüppel. In 100 grammes of dried T.B. he found:—

Fat and wax	26·5 grammes.
Nucleic (tuberculinic) acid	8·5 „
Protamin	24·5 „
Nucleo-proteid	23·0 „
Mineral substances	9.2 „
Proteinoid substance	8·3 „
	100·0 „

Of these, the nucleic acid seems to be important, as according to Behring it possesses in a high degree the specific properties of Koch's tuberculin.

The exact nature of the proteinoid substance is not known, although Rüppel has suggested that it may be of the nature of chitin or some allied body.

This is no doubt a point of great biological importance, as chitin is supposed to be exclusively an animal product, although Wintersteiner has shown that genuine chitin is an important constituent of certain hymenomycetes like Agaricus Campestris.

Recently McLeod and I have investigated this subject, operating on large quantities of tubercle bacilli and timothy grass bacilli. We have so far confirmed Aronson's view that the substance which gives the acid-fast characters to both bacilli is not a fat, but a wax. We have dislodged this substance from the bacilli by Aronson's method, and by other wax solvents like boiling benzene or boiling chloroform. The amount of fat, fatty acids and wax constituted 43·7 per cent. by weight of the dried tubercle bacilli, and 49·0 per cent. by weight of dried bacillus phlei. Besides these bodies we have extracted lipochromes, which in part at any rate give the peculiar colours to these respective bacilli. In its purified condition the tubercular wax is a snow-white powder, which, on being stained with warm carbol-fuchsin, is intensely acid- and alcohol-fast. The removal of fat alone does not affect in the slightest degree either the form or the staining properties of the bacilli. With the removal of the wax, on the contrary, bacillary form is no longer recognisable as such. After removal of fat, wax, proteids and salts, a residue remains which contains nitrogen (3·385 per cent.). In addition to nitrogen the body also contains polysaccharide, which, after hydrolysis, readily reduces an alkaline solution of cupric hydrate. So far we do not feel justified in expressing a definite opinion on the nature of this body, but the probability is that it is of a chitin-like character. In the cultures of timothy grass bacillus a similar substance has been found by us.

One very important problem in the biology of the tubercle bacillus remains

Digitized by Google

to be touched, *viz.*, the question of the existence of spores. The clear spaces in tubercular bacilli can now no longer be looked upon as spores, but are probably degenerative in character, and although other granules have been described by Babes and Czaplewski, there are no facts which show that they are spores, or that the tubercle bacillus in any of its phases possesses a resistance to deleterious agents comparable to genuine endospores.

The main facts, then, to which I have directed especial attention are these:—

1. That under certain conditions the bacillus of tuberculosis assumes forms other than rods. It may be in filaments, or it may show clubs, and there may be a definite actinomycotic arrangement of the fungus.

2. In general the tubercle bacillus is difficult to cultivate, and is not adapted for a saprophytic existence, although it may be got to grow on media of relatively simple constitution.

3. With few exceptions the temperature limits necessary for its growth are high.

4. Under all known conditions the organism is "acid-fast."

5. The cause of the acid-fastness is a body of the nature of a wax.

6. In its chemical constitution the tubercle bacillus contains especially a relatively large amount of fat and wax, and a highly resistant body probably chitinous in nature.

7. There are numbers of acid-fast bacilli other than Koch's bacillus.

8. In the timothy grass bacillus, at any rate, the staining reactions are also referable to wax.

9. These acid-fast non-tubercular bacilli are widely distributed in nature. Some appear to be obligated parasites, others are facultative parasites, others again are saprophytes.

10. The exact relation of these acid-fast bacilli to each other is not definitely known.

11. There is no evidence that genuine endospores are formed by members of this group.

If I might suggest some of the questions which especially require elucidation, I should draw attention to the following:—

1. All facts in regard to the formation of actinomycotic types of tubercle bacilli and its allies.

2. Evidence which would point to the possibility of the extra-corporeal multiplication of the tubercle bacillus and other parasitic members of this group of microbes.

3. Evidence to show that it is possible to transmute the parasitic T.B. into one of the saprophytic forms or *vice versâ*.

4. Does the tubercle bacillus possess chitin as a constituent?

5. Does the nucleic acid of different acid-fast bacilli differ in chemical composition or in action?

6. Is there any positive evidence of the existence of endospores?

Digitized by Google

DISCUSSION.

Professor J. G. ADAMI: Mr. Chairman, Ladies and Gentlemen,—Dr. Moeller and Dr. Bulloch have put the matter so clearly with regard to these bacteria that there is not very much for me to say. But on going up to the Museum and studying the Swithinbank preparations it struck me that one might sound a note much on the lines of the attempts which have been made in America. Looking at all these various cultures of bacteria, obtained from different laboratories, grown upon different media, one does see very, very clearly the absolute need of really standard materials for culture. Until we get those standard materials for culture, until we get the various laboratories working more together than at the present time, I think we must remain in a state of doubt with regard to the relationships, not only of acid-fast bacilli, but all other bacilli. It is impossible for us to come to any conclusion or exact determination until we have more thoroughly agreed to use certain definite formulæ, as far as possible, with regard to our cultures. So I do not think the time is ripe to-day for us, as a section of this Congress, to come to any conclusion with regard to the relationships of these forms. We are only at the beginning of matters; now is the time for us to come together more, and to determine this matter of the regulation of our media, because, looking at Mr. Swithinbank's preparations, one is very much struck by the fact that he has, on his regular media, obtained a constancy. Thus it seems that as the universality of the medium becomes established the apparently aberrant forms come together and grow closer. And so much does this strike one that the conclusion must be forced upon us that we are not ripe to come to any conclusion. We must at the present time, I think, agree to get an international code of media, which shall be employed in one or other direction. More than this I would not say this morning. There are others present who are more directly associated with this subject of acid-fast bacilli, but I would urge as strongly as I can that the time has come for bacteriologists in various lands to obtain standard media, thereby settling to some extent this difficulty of determining relationships.

Another point is with regard to the inoculation. It strikes me that Professor Moeller did not say very much, along the lines of the work done by Professor Nocard, of putting bacteria into animals in capsules, and taking a whole series of rabbits, and trying various bacteria, to see whether any show a closer relationship.

Then testing will give us the best result; and having done that—it will take a year or two to do it—we shall be able to see a little more clearly into this matter of relationships. But the crying matter at the present time in bacteriology seems to be that we are passing through a stage of doubt about relationships, and it is only by the employment of standard media, and by methods such as Nocard's, that we can come to a conclusion and be able to establish definite relationships. (Applause.)

Digitized by Google

Dr. E. J. McWeeney: I had not prepared any remarks upon the subject, but I have been doing some work on these micro-organisms, and I had hoped by the scrutiny of cultivations to have been able to come to some conclusions with regard to the relation of these organisms to each other. I am bound to confess, however, after a study of the organisms and of the various acid-fast organisms—those from the grass, those from butter, and other organisms—they appear to me to be, as Professor Adami states, in a very complex state. The inter-relations of these organisms still require a great deal of work before we can clear the matter up. The relations of them, especially, to genuine streptothrices seem to be particularly close. I have on many occasions remarked the clubbing of the lateral branches of the tubercle bacillus in sputum. After a very careful study of Dr. Coppen-Jones' and Dr. Fisher's papers upon the subject, I am inclined to think that this relation is something more than a superficial one. These clubbings of the lateral branches must have some biological significance, and I am strongly of opinion that as we go on working on these organisms we shall find that biological significance will become better and better marked and understood, and we shall ultimately find, I think, that there is a close relation between the tubercle bacillus, its allies, and the streptothrices. The main difference appears to be that in the acid-fast bacilli there is a coating or sheath of some waxy substance, which seems to be absent from streptothrices. I do not know of a genuine streptothrix in which filaments are present, or where there is a sheath of waxy or fatty material. I just merely make these remarks and throw out these suggestions by way of keeping the debate going until some of the other gentlemen come whose names have been announced from the chair, and others who may like to join in the debate.

Dr. A. Marmorek (from the Institut Pasteur in Paris) attire l'attention sur le fait que le nombre de bacilles qui résistent à l'alcool et aux acides (acid-fast) augmente trop. L'observation des cultures faites au moyen d'un milieu toujours identique, tel que celui exposé par M. Swithinbank, nous prouve que de trop grandes ressemblances existent en ce qui concerne l'aspect extérieur et que par conséquent nous devons nous bien garder d'y baser des distinctions entre tous les bacilles tuberculeux et paratuberculeux. Au contraire, nous devons tâcher de simplifier la question, et en tous cas de nous fier seulement à l'heure actuelle aux lésions anatomiques. Nous pouvons dire que de tous ces microbes seuls sont tuberculeux ceux qui donnent aux petits animaux de laboratoire (cochons d'Inde et lapins) des lésions connues de tuberculose.

Dr. Bousfield: Mr. President, Ladies and Gentlemen,—I should like, with your permission, sir, to say a few words, not because I have anything to add to what has been said as to the particular characteristics by which the various species of acid-fast bacilli are distinguished, but to point out that there is a side of the question to which, perhaps, if I may say so, the gentlemen who have read these papers have hardly done justice. I mean justice from the

Digitized by Google

point of view of those of us who have practically no time for the refined observations to which they have devoted themselves, but who are anxious, above everything else, to have at our disposal the very best tools possible for our daily use in endeavouring to protect the health of the population in general against pathogenic micro-organisms. I admit that the difficulty in a paper of this extent to cover a subject like this is very considerable; but although I have listened with extreme care to both the papers, I am bound to say that at the present moment, notwithstanding that the names of various organisms have been mentioned, I have not heard anything which is calculated to tell me, when examining a deposit or milk suspected of being contaminated with tuberculosis, whether the acid-fast bacillus which I find is the tubercle bacillus; nor is there any indication of the lines along which I must proceed in order to be able to say whether that is so. The remarks which have fallen from subsequent speakers do not make that easier. An examination in this country, to be of use, must be made rapidly, because the Food and Drugs Act requires that proceedings shall be taken within a month. What bacteriologists are sighing for is some method which will enable us to decide in three weeks whether the particular organism found is the tubercle bacillus or not. Nothing less than that will satisfy us. I might, perhaps, be allowed to utter a word of warning to the effect that until some such test is attainable on this question it would be wiser not to say too much about the very great difficulty that there is in recognising the tubercle bacillus in milk. When we come to bring this question forward in the interests of public health we may find it our duty to do so; but then we may be met by the declaration that nobody is able to say whether a given organism is that of tuberculosis or not.

Professor Dr. KOSSEL, *Berlin, Charlottenburg:* Wenn ich einige Worte zu dieser Frage sprechen darf, so möchte ich die Discussion auf den Punkt zurücklenken, von dem sie ausgegangen ist. Ich glaube, es wird noch an anderer Stelle Gelegenheit genug sein, die Frage zu discutieren, zu welcher Gruppe wir die Tuberkelbacillen der Milch zu rechnen haben.

Ich möchte hier nun zunächst mich stützen auf die Untersuchungen, die von Dr. Weber im bakteriologischen Laboratorium des kaiserlichen Gesundheitsamts in Berlin gemacht sind und einen Punkt in den Vordergrund stellen, auf den Dr. Marmorek hingewiesen hat, das ist die Giftigkeit auf die kleinen Versuchstiere, und es wäre mir sehr interessant, hier über die Pathogenität der säurefesten Bacillen näheres zu hören. Es ist interessant, dass Vergiftungen bei den kleinen Versuchstieren nach den säurefesten Bacillen auftreten, wenn sie in grösseren Mengen injiciert werden. Das darf uns nicht wundern. Wir wissen ja, dass alle Bacterienarten in grösseren Mengen pathogenetische Veränderungen hervorrufen, wie auch tote Tuberkelbacillen die ähnlichen Erscheinungen machen wie lebende. Wir müssen uns hüten, Veränderungen, die wir nach Injection von säurefesten Stäbchen bekommen als Veränderungen durch die Tuberculose aufzufassen.

Ich möchte da aufmerksam machen auf die Erfahrungen aus früheren Jahren, dass man Riesenzellen erzeugen kann durch Injection von allen

Digitized by Google

möglichen verschiedenen Substanzen, dass also das Factum, dass nach Injectionen eine Riesenzelle auftritt, uns nicht berechtigt zu sagen, dass eine Tuberkelbacillus sich vorfindet.

Bei den Versuchen des Dr. Webers ist es nicht gelungen, durch Injection von säurefesten Stäbchen verschiedener Herkunft bei den kleinen Versuchstieren eine fortlaufende Krankheit hervorzurufen, wie sie auftritt, wenn man Tuberkelbacillen in kleinen Mengen in die Haut von Kaninchen und Meerschweinchen injiciert. Es wäre mir daher sehr interessant, von den Herren, die über die Säurefestigkeit der Bacterien gesprochen haben, zu hören, ob nach der Injection von säurefesten Stäbchen eine fortlaufende zum Tode führende Erkrankung entstanden ist, welche mit pathologisch-anatomischen Veränderungen wie die Tuberculose einhergeht, in welcher sich auch Bacterien vorfinden in einer Weise wie bei Tuberculose.

Ich möchte von vorn herein sagen, dass gewiss die säurefesten Stäbchen sich im Tierkörper vermehren. Wenn man sie injiciert, so kann man Befunde erhalten, wo die Organe durchwuchert sind, wo sich aber nicht die geringsten Veränderungen finden, die an Tuberculose erinnern, wo die Versuchstiere gar nicht darunter leiden, wenigstens wo man an den Tieren keinen kranken Eindruck gewinnt. Wenn man sie tötet, bemerkt man es; sonst bleiben sie am Leben und gehen nicht, wie es bei Tuberculose der Fall ist, ein.

Es wäre mir sehr interessant, über diese Frage Auskunft zu erhalten (Applause.)

The CHAIRMAN: There is no doubt this is a very difficult subject, and I am afraid it is one which cannot be settled off-hand. It will require a considerable amount of investigation before we can get much further. There is evidently a good deal of work being done, which should throw very considerable light on the life-history of the tubercle bacillus. I think at this stage of the proceedings we must ask those who opened the discussion to add anything that they wish in connection with what they have already said. There may have been some point raised which Dr. Bulloch could answer; and if Dr. Moeller should return to the Section I will ask him if he will say anything further.

Dr. MOELLER, in answer to Dr. Kossel: In the tubercles produced by Koch's tubercle bacilli and the tubercles produced by pseudo-tubercle bacilli there is a visible difference; whilst the genuine tubercles are of a solid proliferating kind, the pseudo-tubercles are of a more inflammatory character with tendency to abscess formation.

Dr. BULLOCH: I have very little in the way of personal experience to add to my remarks. Bacilli were isolated by me from timothy grass which came from Gloucestershire, but I have never succeeded in killing an animal with that when injected into it, though I have introduced large quantities of it. I have seen animals so treated die of inter-current affections, but not of

Digitized by Google

the progressive disease. Five or six guinea-pigs have been killed by it in my hands. But in experiments published by Miller, and experiments done in the University at Copenhagen, it was observed that there was a definitely progressive disease, to such a small extent that with four or five passages of timothy grass through guinea-pigs they could not get it to grow upon ordinary media; as if there were some transplantation going on from the timothy grass, something which is parasitic. I have performed experiments upon mice without definite results. In guinea-pigs there was an abscess at the point of inoculation, but no progressive disease.

The paper which I have read this morning did not deal so much with the differential diagnosis; it dealt with the tubercle bacillus and its allies. With regard to the actual diagnosis, I think the practice of most people shows that the mere microscopical diagnosis of the tubercle bacillus is no longer justifiable. In a court of law it would be impossible to say that it was the tubercle bacillus. The grass bacillus requires a high temperature, and needs a long time to grow, and I think that would be taken as diagnostic evidence that it was the timothy grass bacillus. That was the only point which I thought I would like to add to the remarks I have made. That is the only method by which it is possible to say definitely that the organism is the tubercle bacillus. As I mentioned in the paper, it is not possible to make a merely microscopical diagnosis, but there should be a cultivation from the inoculated animal, and the long period of growth which elapsed and the acid-fast characters should decide the point.

The CHAIRMAN: We have to thank these two gentlemen very heartily indeed for their contribution to the discussion. I think Dr. Bulloch's work has certainly given us valuable information, and opens up wide lines of work.

This concludes the fixed portion of our morning's work. But you will notice that at the end of the programme we have a certain number of papers, and it is desirable that we should distribute these papers over the different days of the Congress. We have with us this morning Dr. Lydia Rabinowitsch, of Berlin, who has consented to give us her paper "On the Infectivity of the Milk of Tuberculous Cows, the Bacteriological Diagnosis, and the practical value of Tuberculin in the extirpation of Tuberculosis among Cattle." I think also that Professor Delépine is here, and Professor Nocard. I hope that Professor Bang will also take part in the discussion.

Dr. LYDIA RABINOWITSCH read her paper, entitled "On the Infectivity of the Milk of Tuberculous Cows, the Bacteriological Diagnosis, and the practical value of Tuberculin in the extirpation of Tuberculosis among Cattle:—"

The frequency and constant increase of tubercular disease among cattle has long ago drawn serious attention to the question whether the milk of tuberculous cows is capable of carrying infection to other animals. By means of minute and varied experiments it has been established beyond doubt that

Digitized by Google

such is indeed the case: tuberculosis can certainly be produced in calves and swine through feeding them with infected milk. Nevertheless it still remained to be shown how very common this mode of infection is under natural conditions. The magnitude of this danger—*viz.*, the spreading of tuberculosis among animals due to the consumption of milk—is best illustrated by the frequent occurrence of tuberculosis among calves, and by the alarming increase of the same disease among swine which has been observed within the last few years. About 90 per cent. of the latter cases have been proved to originate in feeding with infected milk.

The frequent occurrence of intestinal infection need not be wondered at when we consider the extent of tubercular disease among milch cows, as well as the fact that for a considerable length of time after birth the young calves are almost exclusively fed with their mothers' milk. The increase of tuberculosis among swine is further explained by the fact that the young pigs are likewise raised on cows' milk, the principal fodder of pigs in our days of large dairies consisting in the raw dairy residues, made up of the deposits and the lean milk left over from the centrifuging process.

The transmission of tuberculosis among cattle and pigs by means of milk being established, it now remains to be seen whether the milk of all tuberculous cows contains tubercle bacilli, or whether the infectious properties are confined to special forms of tubercular disease.

The investigations of Bollinger, Bang, and a great number of other authors, have established, many years ago, that the milk of cows suffering either with general tuberculosis or with tuberculosis of the udder contains tubercle bacilli. A few observers, such as Hirschberger and others, have experimentally demonstrated the infectious quality of milk derived from cows with incipient tuberculosis and non-tuberculous udders.

Since the important discovery of tuberculin, however, there has been no examination into the question as to whether tubercle bacilli may not be present in the milk of animals which do not show any clinical signs of tuberculosis, but which nevertheless are shown, by their susceptibility to the tuberculin test, to be tuberculous. A positive answer to this question was furnished in 1899 by joint investigation of myself and Dr. Kempner. We found that tubercle bacilli may be present in the milk in cases of tuberculosis, latent, but revealed by the tuberculin test. If, then, the milk of such cows contains tubercle bacilli, it must needs be regarded as infectious, and the consumption of such milk must be held no less dangerous to young animals than that coming from cows with advanced general disease or with diseased udders. In these, as well as in my subsequent experiments, I tested the presence of tubercle bacilli in the milk by means of intra-peritoneal injection on guinea-pigs, as I consider this method to be not merely satisfactory, but, indeed, the only safe method. The alternative way to prove the infectious character of milk, *viz.*, by feeding animals with it, is unreliable, since the quantity of tubercle bacilli thus introduced in the course of one or several experiments is greatly inferior to the amount which the animals would actually consume within a given period of time as part of their food. In the

Digitized by Google

long run even a very slightly infectious kind of milk is bound to produce the disease.

The presence of tubercle bacilli in the milk of cows that respond to the tuberculin test without showing clinical evidence of tuberculosis was proved in the same year by the thorough experiments of Adami and Martin. A further proof in support of this theory was furnished by my last year's observations on milk supplied to infants in Berlin. I found no tubercle bacilli in the milk taken from cows which had been regularly subjected to the tuberculin test, while the supply obtained from animals not subjected to this test contained tubercle bacilli, though they showed no clinical symptoms of tuberculosis.

My own bacteriological observations of former years, as well as the more recent investigations concerning chiefly the milk of cows with suspected udder tuberculosis, established the following facts :—

The detection of tubercle bacilli by means of microscopical examination of cream and sediment specimens of centrifuged milk is unreliable, partly because the absence of acid-resisting bacilli in the examined specimens cannot be considered convincing, as their number may be so small as not to be found in every slide examined under the microscope, whilst even a very small number of tubercle bacilli can be detected by animal experiment. Besides, there occur in milk bacteria which, with regard to their growth, as well as to the effect they have on the animal organs, are very similar to the acid-resisting bacilli which were found by Koch in butter, and which, under the microscope, are unfortunately very difficult to distinguish from tubercle bacilli. These bacilli were not only found by Petri, Beck, Santori, myself, and others in the ordinary market milk, but we were also able to detect and isolate them by animal experiments from specimens of milk obtained from individual cows under aseptic conditions. The above-mentioned discovery of the acid-resisting bacilli, so similar in their nature and appearance to tubercle bacilli, in the milk of cows suspected of tubercular disease, made by our observations some years ago, has been recently confirmed by the experiments of Kühnau. This investigator was able, both in specimens of mixed milk and also in milk taken under aseptic conditions from individual cows, to prove by animal experiment the presence of those bacilli in 20 to 25 per cent. of the cases examined. Whether these bacilli are secreted by the mammary glands, or whether they enter the milk ducts of the frequently inflamed udders from without, is still uncertain, and is for the differential diagnosis of these bacteria without importance. In the light of recent investigations we certainly need not be surprised to find these bacilli in the milk. Already years ago Severin found the same kind of bacteria in cow-dung, while Olt demonstrated their presence in the contents of the intestine of tuberculous as well as of healthy cattle. Moëller further succeeded in obtaining isolated cultures of these acid-resisting bacteria from the cow-dung as well as from the timothy grass used as fodder. The same kind of acid-resisting bacteria, or similar ones, have been recently detected by Cowie on the udder, and by Ludwig Neufeld in the mammary gland of the cow. Besides these acid-resisting bacteria, Nocard

Digitized by Google

and Mollereau, Bang and others, detected, some years ago, saprophytic as well as pathogenic bacteria, esp. streptococci, in the udder of cows suffering from mastitis. These discoveries were recently confirmed by the experiments of Ward as well as by those of Reed and Ward. The last-named authors have even isolated streptococci, along with harmless bacteria, from the milk of cows which were apparently healthy, showing clinically no signs of mastitis. When the udder of the slaughtered animals was examined, streptococci of the same kind were found to be present in all parts of it. The other bacteria, too, which were found in the udder after the cow was slaughtered, proved identical with those previously isolated from the milk. That streptococci may be present in the milk a long time after the mastitis has disappeared has recently been proved by the experiments of Lameris and van Harrevelt.

In view of these facts, the presence in cows' milk of the bacteria which resemble tubercle bacilli is, after all, not difficult to explain, as these microbes, as well as streptococci, can either enter the udder from the outer world through the teat, or they can be taken in with the food and absorbed by the blood and lymph-stream from the intestinal canal, and carried thence to the udder. In the present state of our knowledge it is not yet certain whether these bacteria can be the cause of the mastitis, or whether the mastitis existed already previously, the inflamed tissue furnishing a suitable nourishing medium for the development of the microbes. As these acid-resisting bacilli are sometimes visible in clusters in a specimen of centrifuged milk, a microscopical differentiation of these from the genuine tubercle bacilli, especially when both kinds are present mixed in one and the same specimen of milk, is rendered extremely difficult or even impossible.

The only and the safest way to prove the presence of tubercle bacilli in milk is, and remains, therefore, the animal experiment, namely, the intra-peritoneal injection into guinea-pigs of a mixture of the cream and sediment of thoroughly centrifuged milk. The following interesting experiment made by Ostertag shows how very susceptible the guinea-pig is when subjected to the intra-peritoneal inoculation. The milk of a cow with tuberculous udder which, when 20 ccm. of it were given through the mouth to a guinea-pig, produced intestinal tuberculosis, was able to produce general tuberculosis when injected into the peritoneal cavity of guinea-pigs, even after it had been diluted a hundred thousand times. By comparative experiments I was, indeed, able to prove that the last milk obtainable from the udders of suspected cows gave positive results in regard to the presence of bacilli, whilst the first milk obtained from the full udders was not able to convey tuberculous infection in the animals.

It must further be noted that it is necessary to examine the suspected milk repeatedly, and at various times, as a single negative result cannot be considered finally convincing. Although it is easy to prove the presence of a small number of tubercle bacilli by means of inoculation, the result of the experiments may be obscured in cases where changes are produced in the tissues resembling tubercle when examined with the naked eye, but which in

Digitized by Google

reality have been caused by the action of the above-mentioned acid-resisting bacilli. Their pseudo-tubercular nature can easily be proved when examined under the microscope. But, as we have already seen, real tubercle bacilli may be found in the tissues along with the acid-resisting bacilli; it is, therefore, advisable to carry on, in addition to a histological examination, inoculation experiments with parts of the suspected tissues, until positive results are obtained. Thus the process of diagnosing tubercle bacilli by means of experiments on animals may become very tedious, and two or even three months may elapse before we are able to decide whether the milk of the cow suspected of tuberculosis really contains tubercle bacilli.

The presence of tubercle bacilli in the milk does not as a rule correspond to the extent of the disease in the animal, as we had already occasion to mention. It must be admitted, though, that in cases of extensive general tuberculosis or udder tuberculosis the presence of tubercle bacilli in the milk may be looked for. How very often the clinical diagnosis of udder tuberculosis meets with great difficulties, even in the hands of the most competent observers, I had an opportunity to convince myself in the course of my last year's observations. The milk of the cows in question, as well as the results of the clinical examination, were kindly placed at my disposal by Dr. Schütz, Professor at the Veterinary School in Berlin, and Dr. Schroeder.

I made it my task this time to carry on my experiments on the occurrence of tubercle bacilli in milk only of such cows as have been proved by repeated clinical examination to be suspicious or to show udder tuberculosis. These ten cows, the milk of which was repeatedly examined microscopically and by animal experiment, were not submitted to the tuberculin test. In our this year's experiments, we merely wished to ascertain whether the clinical examination of a cow, especially of her udder, allows the inference that the milk of the animal is infectious.

It would lead us too far to enter here into details of our experiments. I shall only mention the results of the examination of those cows which throw most light on the subject in question.

In one case, where the clinical diagnosis pronounced the udder to be tubercular, we detected the presence of tubercle bacilli only with the aid of animal experiment, and failed to do so with the microscope. In another cow with the same diagnosis the microscopical examination of the milk repeatedly revealed a great number of acid-resisting bacilli, while the animal experiment gave negative results. The autopsy of this cow did not confirm the clinical diagnosis of tuberculosis of the udder; it revealed merely the presence of chronic interstitial mastitis. Inoculation experiments with portions of the suspected udder also failed.

In two other cows, in one of which the diagnosis of udder tuberculosis was made with great certainty, while in the other a strong suspicion was entertained, microscopic examination repeatedly detected in the milk a great number of acid-resisting bacteria, of the nature of which we could not be sure: we thought them at times to be real tubercle bacilli, at other times they seemed more like rod-shaped bacilli merely resembling tubercle bacilli.

Digitized by Google

Inoculation experiments with the milk of these two cows produced in the animals experimented upon suspicious changes, which were due to acid-resisting bacilli resembling the tubercle bacilli. Further inoculation with suspected tissues of these animals gave a negative result.

Of special interest was a case where the clinical diagnosis of udder-tuberculosis was confirmed by the post-mortem examination. Neither the microscope nor the experiment on animals could show the presence of tubercle bacilli in the milk. The microscopical examination of sections of the tuberculous udder revealed numerous clumps of tubercle bacilli in the tuberculous interstitial tissue, while there were none to be found in the milk ducts.

From a review of these cases we arrive at the conclusion that the clinical diagnosis of tuberculosis, especially of udder tuberculosis, does not enable us to judge whether tubercle bacilli are secreted along with the milk of the cow in question. For, on the one hand, our previous investigations, as is seen in the protocol of the same, have shown tuberculous disease of the udder sometimes to escape detection altogether, or simple inflammation may be diagnosed clinically, while later on the histological examination, as well as the results of inoculation experiments with the milk and with portions of udder tissue, prove that it was clearly a case of udder tuberculosis. Delépine arrived at the same result in the examination of a cow whose udder was not found to be tuberculous till examined histologically. The milk of this cow contained tubercle bacilli, although repeated clinical examination, as well as the observation of the udder with the naked eye after the autopsy, yielded no grounds for suspicion of tubercular disease. Of course, it is not to be wondered at that the commencement of udder disease escapes detection when palpation is used.

On the other hand, the udder may be clinically pronounced tubercular, while both the autopsy as well as the histological examination clearly show that we have merely to deal with a case of interstitial mastitis or of cyst formation in the udder. This fact was not only proved by ourselves, as may be seen from the above synopsis, but the investigations of Adami and Martin, as well as those of Delépine, illustrated by a series of interesting experiments, led to the same results.

Kühnau, in his recent article on the "Extermination of Tuberculosis among Cattle," has further shown that streptococci, which not infrequently invade the udder, as seen before, are occasionally capable of producing changes closely resembling tuberculosis.

A third possibility has been also demonstrated in the present year by my own investigations, in which I proved that even repeated inoculation of the milk may fail to show the presence of tubercle bacilli, though the clinical diagnosis of udder-tuberculosis was confirmed by the autopsy, and though great numbers of tubercle bacilli could be detected with the microscope in the tuberculous interstitial tissue (though not in the milk ducts).

The presence or absence of tubercle bacilli in the milk of cows with udder tuberculosis is, in our opinion, greatly dependent on the extent of the

Digitized by Google

disease in the organ. It is easy to understand that in a case where the tubercular foci in the udder are circumscribed and encapsuled, the tubercle bacilli will not be able to enter the milk ducts with the same ease as they may do when the disease of the organ is diffused. From examination with the naked eye and under the microscope, as well as histologically, of a number of tubercular udders which were kindly placed at my disposal by Dr. Reissmann, director of the Berlin slaughter-houses, I was enabled to convince myself how very much the occurrence of tubercle bacilli in the milk depends on the extent and the local seat of the tubercular changes in the udder tissue. In many cases of far advanced disease I was only able to detect the presence of tubercle bacilli in the fluid expressed from the milk ducts after having examined a great number of specimens. In several cases I failed to detect tubercle bacilli in the capillary milk ducts under the microscope, while in other cases the milk capillaries were crowded with bacilli.

In view of the great variety of bacilli found in tuberculous udders, as seen in my investigations in this field, I venture to express a doubt whether the method (first recommended by Nocard) of harpooning does always furnish us with a sure means of judging whether in a doubtful case we are dealing with udder tuberculosis or not. Besides this, the harpooning method has been pronounced recently by Kühnau not quite without danger to the animal.

So far we have seen that the clinical examination of milch cows bearing on the question of the detection of tubercle bacilli in the milk is, without the aid of auxiliary methods of research, both insufficient and unreliable. On the other hand, the method of demonstrating the presence of tubercle bacilli by means of experiments on animals may prove very tedious. There remains only the tuberculin test, which is the sole quick and sure method. The tuberculin test is recommended by most of the distinguished authors, along with the clinical examination, as the best means of diagnosing and thus aiding the extermination of tuberculosis among cattle. It is, on the other hand, not difficult to understand that, in view of the enormous spread of bovine tuberculosis, the farmers try to discredit tuberculin. But the great diagnostic value of tuberculin can scarcely be diminished by the fact that the failures in the diagnoses only amount to 2·9 per cent. (as Schütz did prove) of all the cases subjected to this test.

In cases where the diagnosis of tuberculosis has been made with great certainty by means of clinical examination, the tuberculin test becomes naturally superfluous. This does away with the argument that about 10 per cent. of the cattle which fail to react to tuberculin nevertheless are tuberculous. These 10 per cent., as is well known, consist of highly tuberculous animals, which have themselves produced and hold in their organism so much tuberculin that they are unable to respond any longer to the tuberculin test; nevertheless this argument is brought forward again and again by the adversaries of our method. The tuberculin test should only be applied to animals which are suspected of tuberculosis; and, when thus used, failures in the diagnosis never occur, as I am also informed by Nocard, who based his opinion on a prolonged experience in France. Bang, in his last year's

Digitized by Google

treatise, also appears as a champion of tuberculin; for diagnostic purposes he still considers it to be the finest and safest means for diagnosing tuberculosis in animals, in spite of the many opinions to the contrary.

We must point out the fact that the present scientific data with regard to the extermination of bovine tuberculosis clearly show this to be practically impossible without the aid of tuberculin. Naturally we must, in the first place, make a point of exterminating the cows affected with udder tuberculosis and general tuberculosis, since their milk is, more than all other, to be regarded as infectious. In the second place come the animals in which the clinical diagnosis can only be established by the help of tuberculin. These cows must first of all be separated from the healthy ones. The question whether they should be killed, and the proper time for doing so, will depend on the quicker or slower progress of the tuberculous disease. That is to say that, of course, not all the animals which react to tuberculin need be immediately slaughtered. Therefore we may say that, combined with the clinical and bacteriological examinations, the tuberculin test furnishes us with the safest means of obtaining milk free from tubercle bacilli as well as of rearing cattle free from tuberculosis.

The CHAIRMAN: We have a paper on a similar subject by Dr. McWeeney, who is ready to give it to us now, and I will therefore ask him to do so.

(The Chair was temporarily occupied by Professor Hamilton.)

DR. MCWEENEY then made a communication, entitled "On the Infectivity of the Milk of Cows that react to the Tuberculin test in the absence of Clinical Evidence of Tuberculosis." He said:—

In agreement with Dr. Rabinowitsch, I selected the last litre of milk that came from the udder. It was received in a sterile vessel, and poured into a large sterile funnel provided with a rubber tube and burette-clip. The funnel, with its contents covered with a sterile glass plate, was allowed to stand for a day in a cool dairy, and the burette-clip was then opened so as to allow some 50 cc. of the milk, containing all the sediment, to flow out into centrifuge-glasses. After centrifuging, the solid sediment was taken up with wide-mouth pipettes, suspended in about 1 cc. of the milk, and injected into the subcutaneous tissue of the thigh of a guinea-pig. About a cc. of the cream was in some cases injected into the subcutaneous tissue of the opposite thigh. Up to the present I have operated in this way on sixteen animals, and in not a single case have I obtained a positive result. All the animals proved quite free from tubercle at the autopsy, and, the milk being collected with proper precaution, I lost none from sepsis. The milk of five cows that seemed in perfect health, and certainly had no sign of udder-tubercle, was tested, as I have said, with totally negative results. At the autopsy of the cows, I was able to verify the fact that they were slightly affected with tubercle, the mediastinal glands being affected in all, in two the lungs, and in one the lungs, glands and liver. With the object of ascertaining whether very small numbers of tubercle bacilli

Digitized by Google

could really be detected in milk in this way, I added to about a pint of milk some ground-up caseous matter from the cervical gland of a scrofulous child operated on at the hospital. I had failed to find tubercle bacilli in this material. The milk was worked up as above described, and the guinea-pig that was injected developed within a month a caseous abscess in the thigh. The pus contained the tubercle bacillus in moderate numbers. My other control experiment was made with some pus from a tubercular abscess in the lung of one of the cows (No. 17) with milk of which I had been experimenting. This pus must have contained very few tubercle bacilli, for I had failed to find a single one in three or four slide preparations. As much of the viscid pus as would fit on the blade of a scalpel was taken up on a sterile knife, and blended up in about a pint of milk, which was then treated as above described, with the result that the animal inoculated with the sediment of that milk became tuberculous.

I quite admit that the number of experiments hitherto made—only sixteen, four of the five cows having been three times tested and the fifth four times—is quite insufficient to enable one to draw well-grounded conclusions. But this I will say, that I am beginning to be of opinion that, in the absence of tuberculous mammitis, one does not find the milk of tuberculous cows to be infective. My idea is that in cases where the injection of milk from cows, that are *not* the subjects of tuberculous mammitis, *does* produce tuberculosis in the experimental animals, it does so *through infection of the milk by means of dust, vessels, or in some other way after it has left the cow.* I am of opinion that if the milk is collected with the proper precautions against contamination the results of inoculation will prove negative; whereas if one uses milk collected as usual in dairies, and which is the product of several cows, then, out of a large number of experiments, a certain number of positive results will be obtained, and be, as I think, erroneously attributed to the yielding of infected milk by cows in the absence of tuberculous mammitis. I am well aware that in saying this I am placing myself in opposition to other workers, notably to Adami and Martin, but in view of the results which I have obtained, and which are uniformly negative, I must, for the present at any rate, adhere to my opinion. It is my intention to continue this series of experiments, and should the ultimate result fail to confirm my present idea, I shall not fail to publish it.

M. Nocard appuye les conclusions de Dr. Lydia Rabinowitsch. Il n'est cependant pas d'accord avec lui sur tous les points. Le lait produit par une vache atteinte de tuberculose mammaire est très dangereux pour les veaux qui le boivent; mais la mammite tuberculeuse est rare, à peine 1 à 2 pour cent des vaches *tuberculeuses;* en sorte que, là où les veaux têtent leurs mères, très peu deviennent tuberculeux par cette voie. Au contraire, c'est la grande cause des progrès de la tuberculose du porc, dans tous les pays où l'industrie du lait est très prospère, et où l'on rend aux fermiers les résidus de la fabrication du beurre et du fromage. Il suffit d'une seule ferme renfermant une vache à tuberculose mammaire pour infecter le petit lait distribué à

Digitized by Google

toutes les autres fermes. Je suis aussi d'accord avec Madame Rabinowitsch sur la question du diagnostic. Le meilleur moyen, et le plus sûr, c'est l'injection, dans le péritoine du cobaye, du dépôt obtenu par la centrifugation d'une quantité notable du lait. J'y ajouterai le procédé du *harponnage*, qui permet d'avoir un petit fragment du point induré de la mamelle ou du ganglion suspect, grâce auquel ou peut faire le diagnostic en quelques minutes par examen de préparations par frottés.

Enfin je demande à Madame Rabinowitsch la permission de lui signaler une petite omission : le danger du lait tuberculeux n'est pas limité aux veaux et aux porcs ; il est surtout grave pour les enfants qui sont nourris exclusivement de lait de vache. Nombreux et très connus sont les faits cliniques qui le prouvent. Celui recueilli par le docteur Gosse (de Genève) sur l'une de ses filles a presque la valeur d'une expérience. Enfin les travaux du grand hygiéniste anglais Thorne-Thorne le prouvent surabondamment. Alors qu'en Angleterre la mortalité générale par tuberculose a diminué de 45 pour cent en 50 ans, la mortalité par tuberculose abdominale des enfants âgés de moins d'un an a augmenté de 27 pour cent.

"C'est que," dit Thorne-Thorne, "tous nos efforts ont été dirigés vers l'assainissement de la maison, de l'atelier, de la ville, diminuant ainsi les chances d'infection par les voies respiratoires, les plus redoutables pour les adultes ; nous n'avons rien fait pour éviter l'infection par les voies digestives la plus fréquente chez les enfants ; nous n'avons rien fait pour assurer l'état sanitaire des vaches laitières."

Tous ceux qui ont étudié la question seront de son avis.

(Dr. Sims Woodhead resumed the Presidency.)

Professor J. G. ADAMI, M.D. : Mr. President, Ladies and Gentlemen,—There are rumours going about that this afternoon there is to be another example of Balaam brought before us, and possibly our discussion on the lines which it is taking is a little premature. There is no harm in that, but this is an enormous subject which has been brought before us, and one is nervous as to how to treat it, because one has already heard sounded the pathetic note by Dr. Bousfield as to what the poor State hygienist is to do with regard to this bacteriology, especially that of the tubercle bacillus, and the dangers which are looming before him in relation to city, State, and town councils as to what is and what is not infectious milk. We are in a bad state at the present time, a state which requires nerve, and, above all, truth in these matters—(applause)—so that we must say exactly what we mean. Now, I am in doubt as to how far one ought to discuss the subject which Dr. Nocard brought forward, the general subject of infectivity by milk. But there are some words connected with my personal experience which I might give, such as those given by Dr. Rabinowitsch. Dr. Martin and I at Montreal conducted individual observations along the same lines as she did, and got similar results to those she obtained, and results contrary to those of Dr. McWeeney ; and if Dr. McWeeney will allow me to put out my coat,

Digitized by Google

tails I would say that it was very wrong of him to suggest that the milk in these cases mentioned by Dr. Rabinowitsch was collected in dirty vessels, and that the bacilli got into the milk because we did not employ sterilised vessels and take all precautions. We first used a sterile funnel, and later on our servant got so skilful and so well versed in the matter that he always received the milk direct from the cow into the sterilised flask, without the intervention of any funnel. We found a certain proportion of cases in the ten animals we used, none of which showed any clinical signs of tuberculosis of the udder. I am weak at remembering figures, but out of about one hundred rabbits and guinea-pigs which were inoculated we got two rabbits and three guinea-pigs which did get peritoneal tuberculosis from the inoculation of milk which was centrifugalised before inoculation. It was, I am sure, a very small number. We had to centrifugalise a large quantity of milk to get the results; and when we killed these animals we found—after a year—that none of them showed any obvious sign to the naked eye, or under the microscope, of tuberculosis of the udder. But I must say—and I think Dr. Rabinowitsch has noticed the same thing—that in a large number of animals showing no sign of tuberculosis, and which on section showed no tubercle bacilli, we got a certain amount of indurative mammitis, which was remarkable. On this I am keeping an open mind at the present time. It is remarkable that in those cases where one does get, by examining the milk bacteriologically, a certain rare number of tubercle bacilli and finds no definite tuberculosis in the lymphatic glands, one does get this mammitis present. It was very interesting that in the few cases from these cattle in which we had, by bacteriological examination, found tubercle bacilli, on making a post-mortem we found the disease furthest advanced in the lungs. And so Dr. Martin and I came to the conclusion that there should be a certain amount of examination in cases in which there was no udder-tuberculosis. In none of these cases did we get our bacilli in clumps; we found that they were of very rare occurrence, and it took a long time to get them out, so much so that my laboratory assistant had some affection of his eyes, and I went over every one that he thought he had found. Those found were stunted, but they were acid-fast. But in several cases we did get this disease. I was forced to conclude that under certain conditions we may have bacilli in the milk in cases in which there is no definite tuberculosis of the udder, though, as I say, there may be a certain amount of indurative mammitis.

Turning now to the poor State and municipal bacteriologist and adviser, these results, I must confess, do look bad. One has to say again, "Here is one of those conditions in which you cannot make an absolute diagnosis." However, I would bring a word of comfort here. While I believe it to be true that tubercle bacilli can pass into the milk where there is no tuberculosis of the udder, yet when you find that condition the bacilli are so few and so rare that to all intents and purposes the milk is harmless, because of the very attenuated form in which the tubercular poison is present. In such a case, taking a susceptible animal like the guinea-pig, and injecting large quantities under the peritoneum, it is only very rarely that one produces tuberculosis by

Digitized by Google

that means. So if you take a more resistant organism, like man, the danger from such a source is extremely slight. Still there is a slight element of danger there. On the other hand, for all practical purposes, where you have tuberculosis of the udder you have enormous quantities—millions—of tubercle bacilli, which may be poured into the milk, and that milk is, relatively, highly dangerous. And so, for practical purposes, I confess I agree with you, sir, and others who have written on the subject, that where there is tuberculosis of the mammary gland there is great danger. One speaks, however, with a feeling of waiting to hear what will be said on the subject this afternoon. I agree with Dr. Nocard that there is danger in these undoubted cases in which the milk of cattle has certainly caused tuberculosis in animals.

Lastly, I would only say one word, and that is: we have to remember all through, in discussing this subject of the infectivity of milk, that the mere existence of the bacillus is not the dangerous element; we have constantly to bear in mind that other condition, namely, the susceptibility of the individual. We are too apt to think that if we have a bacillus, then all is up and that it is terribly dangerous; and we are too liable to forget that the virulence of the bacillus is onething to take into account, and another is the susceptibility of the individual. And I think we must be prepared to acknowledge that healthy individuals can take considerable quantities of tuberculous milk without harm. The great danger is in the case of children and susceptible people. It is for them we have got our regulations, and whatever is said this afternoon must be taken in the light of that; we have to deal with these defenceless children. Everything must be done to stop the taking of milk if even it contains one single tubercle bacillus.

Professor HAMILTON, *Aberdeen*: Mr. President, Ladies and Gentlemen,—I have listened with very great pleasure to the discussion upon this extremely important subject as to the presence or non-presence of tubercle bacilli in the milk of animals suffering either from tuberculosis of the udder, or not suffering from tuberculosis of the udder, but from tuberculosis of other organs. It seems to me there is a point which has not been exactly brought forth with the prominence which one would like to see given to it, and one on which perhaps Professor Nocard and others who are engaged in veterinary practice and agricultural affairs may possibly be able to give us some more definite information—I mean as to whether a non-tuberculous calf becomes tuberculous when suckled by a tuberculous mother, whose udder is, however, free from tubercular deposit. That is one of the best experiments which can be made under the circumstances. I may say, for my own part, that my colleague Mr. Wilson, Lecturer upon Agriculture, has shown me a number of statistics giving many years' experience upon a tubercular farm; and the result of that experience is this—that if the mother does not suffer from actual tubercle of the udder, but is the subject of tubercular disease elsewhere, the calf remains free. I do not know whether there are any others in this meeting who have had a similar experience, but it would be extremely important if we could elicit some facts bearing upon the subject

Digitized by Google

Dr. M. P. RAVENEL, *Philadelphia*: I am like Dr. Adami and other gentlemen who take umbrage in a mild way at what Dr. McWeeney says about the non-sterile vessels. In a series of experiments conducted by the Department of Public Health in Philadelphia in 1898, I gave the results of researches carried out by the Sanitary Board of Pennsylvania on five animals, none of which showed tuberculosis, either *ante mortem* or *post mortem*, although in only one of these was a histological section made. Over 400 guinea-pigs were inoculated with this product, which was centrifugalised, and of these 400 guinea-pigs $10\frac{5}{12}$ per cent. showed tuberculosis.

So we are very strongly of the opinion that in a certain number of cases at least the milk of cows which show no evidence of udder tuberculosis, but which react to tuberculin, unquestionably can contain virulent tubercle bacilli.

The number of tubercle bacilli varied from time to time in the series of experiments, which extended over a considerable length of time. In regard to the poor health authority, it may be interesting to some of you to hear what we do in the United States of America.

We have no central government which controls these matters; each State, and oftentimes each municipality, has its own rules.

In the State of Pennsylvania we have an organised State Live Stock Sanitary Board, and we have in Philadelphia the inspection of what we speak of as "notified milk," the Walker Gordon Laboratories, of which there is a branch in London, being one of the best of these.

In our examination of the milk for these dairies, we depend entirely on the tuberculin test. We make inoculations of animals with centrifugalised milk, and if we get positive results we say that that milk is unfit for drinking. But we cannot wait for that test, so we have at least monthly or bi-monthly inspections which embrace every cow in the dairy by a veterinarian, and at least two tuberculin tests are made during the course of the year. That is what we depend upon in order to keep the milk good.

Dr. BOUSFIELD: Mr. President, Ladies and Gentlemen,—I should like to ask one question which has suggested itself to my mind in connection with every speaker up to the present, namely, what proof there is—and I have not heard any advanced—in the case of animals which react to tuberculin, in which there is no tuberculosis of the udder, that there is no tuberculosis; on what do they base their assumption that there is no tuberculosis? It seems to me, in the cases mentioned by Dr. McWeeney as being due to contamination of the air, that the twos and threes of tubercle bacilli are due to the extremely small amount of tubercular disease present, which was not detectable by any of the means at our disposal.

The CHAIRMAN: Before Dr. Rabinowitsch replies, I should like to make one or two observations upon this very important subject. Corporation bacteriologists must be content to use such methods as are at present at our disposal. Those who have followed the work of this Section must see that

Digitized by Google

there are a number of difficulties which have to be faced and questions which have to be solved yet. We must not go ahead too rapidly and try to put into practice methods which are not yet thoroughly reliable, or at any rate which our knowledge does not justify us in relying upon too greatly.

There is one very safe rule in connection with all this, namely, that we shall always err on the safe side. That, indeed, in this matter, is a very important point. There is one question which Dr. Rabinowitsch brought forward, which I think is of very great importance indeed, and that is the method of experimentation. I was very glad indeed to hear that she used almost entirely the intra-peritoneal injection. There is no doubt that much more reliable and more accurate results are obtained by that method than by any feeding method which has yet been devised. We know as a matter of fact that only in a very small proportion of cases in which there are undoubted tubercle bacilli in the milk could we rely upon experiments carried out by feeding. I have had some experience in that matter, and I am glad that it has been brought forward so prominently. There is another point which we must consider, namely, that in connection with this Dr. Rabinowitsch was insisting very strongly on the use of tuberculin. There are difficulties about the diagnosis even with all the assistance which one can obtain from trochars and the like, difficulties in connection with the diagnosis of tuberculosis of the udder, which are exceedingly great.

I remember seeing three tuberculous udders on the table at one time, in not one of which tuberculosis had been diagnosed, even by very able veterinary surgeons. These instances were brought forward to show the very great difficulties which exist in the diagnosis of tuberculosis of the udder, even when one knew that there was tuberculosis in other parts of the body. In these cases there were certain small ulcerating areas and a little hardness. The ducts were opened into a good deal, and the tuberculosis was found to be entirely confined to those ducts, and, moreover, the ducts in the central part of the udder. In such a case it is almost impossible to determine that there was tuberculosis in that position. One of the great advantages, as pointed out by Dr. Rabinowitsch, that we have in tuberculin is that it is an agent which will undoubtedly tell us in these cases of moderate tuberculosis whether we are really dealing with tuberculosis or not. If there is any lesion of the udder of any kind, it does not matter whether we are able to make out whether it is tuberculous or not, but where there is even any suspicion of lesion of the udder, if there is a reaction to the tuberculin I hold that all the bacteriological examination in the world should not convince one of us that that milk should ever be used. That is a point which we should insist upon strongly, and it is one also which may carry some comfort to the mind of the medical officer of health. This is the real question at issue.

There is another point that I should like to mention, as I have examined a considerable number of cases of tuberculosis in children. To my mind there is no doubt that the glands of the neck are infected, in a considerable proportion of surgical cases of tuberculosis, from the milk, that is to say, milk given to young children. I cannot give the data on which I have founded

Digitized by Google

that opinion, but it is my firm conviction that a number of these cases of so-called scrofulous glands of the neck are the result of infection of the glands by tuberculous material, the original seat of invasion being the tonsil. I have followed this very carefully in pigs, and equally carefully in children, and I wish simply to state that that is my strong conviction. I shall ask Dr. McWeeney to reply, and perhaps Dr. Rabinowitsch will kindly close the discussion.

Dr. McWeeney: Mr. President, Ladies and Gentlemen,—It is only right to say that my views on this subject have been to some extent altered by the debate which I have just heard.

I admit that it is not safe to assume that an udder is non-tuberculous because by no veterinary examination can the fact be detected that it is tuberculous. It would appear that even when one is making a microscopical examination, if you find a certain amount of interstitial adenitis, there is a great possibility that that apparently non-specific adenitis may be really of tuberculous origin.

I have had a series of negative results, but I am free to admit that my experiments have not been sufficiently numerous to enable me to form a very decisive opinion upon the matter.

With regard to the value of tuberculin tests as a diagnostic where, in the bovine animal, tuberculosis exists, I express myself completely in accord with the views stated by Dr. Rabinowitsch and other speakers. When the animal reacts to tuberculin, if I fail to find a tubercular lesion I do not any longer assume that the tuberculin test is wrong, but rather conclude that my examination has been incomplete.

The Chairman: It appears that Dr. Rabinowitsch is not inclined to reply, but I think you will agree with me that we should like to hear the views of Professor Bang. He and Professor Nocard are to a great extent our guides in this matter. Perhaps Professor Bang will be good enough to close the discussion instead of Dr. Rabinowitsch.

Professor Bang: Gentlemen,—I am sorry I have not been able to hear what has just been said in this Section, thus it is rather difficult for me to accept the invitation of the chairman to take part in the discussion. I only know, through what Professor Woodhead has kindly told me, that you have been speaking about tubercle bacilli existing in the milk without inflammation of the udder. On this question I have made many experiments years ago, and therefore I may perhaps call attention to them. I have found that it is very rare to find tubercle bacilli in the milk of a cow which has not tubercular lesions of the udder. Nevertheless, it does sometimes happen. Many years ago I published a paper here in London at the Congress on Hygiene, and in that I said that out of sixty-three cases of cows with advanced general tuberculosis, but with healthy udders, I was only able to produce tuberculosis in guinea-pigs by the injection of the milk of nine of those cows. And in

Digitized by Google

some of these cases there were some very small nodules in the udder, although there were no clinical appearances of the disease. In one case at least I had to deal with acute miliary tuberculosis, because tubercle bacilli had been carried through the whole body. But there were a few cases where I had only very advanced tuberculosis, without any alteration to be found in the udder. Therefore I think that it happens very rarely that tubercle bacilli on such occasions are discharged with the milk; so rare is it that it has no practical value, I think.

Another question under discussion, as I understand, was as to the value of tuberculin for diagnosis in such cases. I am not of the opinion that has been expressed here. Some people think, as far as I understand it—and especially I have heard it in the Veterinary Section—that if they have to do with a case of enlargement of the udder, or inflammation of the udder, which does not seem to be tuberculous, they have not the clinical symptoms of tuberculosis in the udder, and they are not able to find tubercle bacilli in the milk. But if they inject tuberculin and the cow reacts, then they believe that it is a case of tuberculosis in the udder. Am I right? (Yes.) Well, this conclusion I find to be illogical, because you know that a very, very great number of cows will react if you inject tuberculin into them, and they react because they have tuberculous affection in some part of the body, mostly in the lymphatic glands, in the bronchial glands, or elsewhere. If such a cow has benign inflammation in the udder there is no reason to think that it has a tubercular inflammation of the udder, because the cow reacts. A cow which has tuberculosis in the glands may have, at the same time, a non-tubercular affection of the udder, and therefore this diagnosis is of no value.

(The Section then adjourned.)

Digitized by Google

THURSDAY, JULY 25th, 1901.

PROFESSOR SIMS WOODHEAD, M.D., F.R.S.E., *President of Section*, IN THE CHAIR.

The PRESIDENT: We have to-day a discussion on "The Varieties of Tuberculosis (Morbid Anatomy and Histology)." We have amongst us Professor Benda, who has very kindly consented to open the discussion. We hoped Professor Sheridan Delépine, of Manchester, would be here also to take part. His experience would be very valuable to us; but, unfortunately, he is unable to be present. We have with us Professor D. J. Hamilton, of Aberdeen, who will follow, and we hope a number of other gentlemen will join in the debate. I will now ask Professor Benda to open the discussion.

Professor BENDA, *Berlin*: LADIES AND GENTLEMEN, MR. PRESIDENT,—Es ist ein grosses Gebiet, welches hier zur Discussion gestellt ist. Es giebt kaum eine Frage der Bacteriologie und der Pathologie, welche sich nicht in irgend eine Beziehung zur *Variation der Tuberculose* setzen lässt. Ich habe daher eine Auswahl treffen müssen, um nur einen kleinen Teil dieses Gebietes herauszuschneiden, der sich für eine Discussion eignen könnte, und ich glaube, dass das Comite gerade auch im Auge gehabt hätte, dass ich über die Dinge spreche, über die meine Publikationen der letzten Jahre ergangen sind. Ich habe mein Thema insofern erweitert, als ich im Allgemeinen die Variation der *Ausbreitungswege der Tuberculose* in dem inficierten Organismus als Grundlage meiner Besprechung gewählt habe, um an der Besprechung der Ausbreitungswege zu zeigen, dass sie einen wichtigen Bestandteil für eine Variation des Bildes der Tuberculose abgeben.

Der Tuberkelbacillus selbst ist unbeweglich; er hat ein langsames Wachstum und erregt schwere Reactionen an den Stellen, wo er sich festsetzt. Diese drei Bedingungen scheinen dazu bestimmt, um den Organismus gegen den Tuberkelbacillus zu schützen, um diejenigen Erscheinungen, die durch den Tuberkelbacillus hervorgerufen werden, zu lokalisieren, und so sehen wir in der That dass der Krankheitsprocess, wo der Tuberkelbacillus, beraubt derjenigen Gelegenheiten, die ihn passiv weiterverbreiten, nur auf sein actives Wachstum angewiesen ist, lokalisiert bleibt. Ich erinnere daran, dass z. B. die Tuberculose der Lymphdrüsen lokalisiert bleibt, dass viele der grossen Drüsen, Leber, Nieren, Lungen, kleine begrenzte Tuberculosen besitzen können, weil an diesen Stellen der Tuberkelbacillus nur durch die Continuität die Nachbarstellen inficieren kann und weil er es nur höchst langsam thut.

Da wo wir die Ausbreitung der Tuberculose im Organismus gewaltsam verderblich vorschreiten sehen, lassen sich immer besondere Gelegenheiten nachweisen, die den Tuberkelbacillus passiv verbreiten. Das hat sehr wohl

Digitized by Google

bereits der Entdecker des Tuberkelbacillus, Herr Koch, erkannt, der die Verbreitung des Tuberkelbacillus wesentlich auf solche passive Einflüsse, auf die Verschleppung durch Wanderzellen zurückführt. Aber ausser den Wanderzellen sind es die Wege des Organismus selber, die dem Tuberkelbacillus Gelegenheit zur Verbreitung geben, und wir sehen, dass alle Wege, die der Organismus besitzt, vom Tuberkelbacillus zur Verbreitung benützt werden.

Schon wenn wir die Thatsache ins Auge fassen, dass die grossen epithelialen Organe, die Lunge, die Nieren die Stellen sind, wo die grösste Massenentwickelung der Tuberculose beim Menschen vor sich geht, haben wir den Hinweis, dass es die Epithelkanäle dieser Organe sind, die für die Ausbreitung der Tuberculose eine wichtige Rolle spielen. In der That sehen wir, dass wie auch immer diese Organe inficiert werden, ob sie primär inficiert werden oder ob sie secundär oder metastatisch aus dem Organismus inficiert werden, sofort die epithelialen Teile dieser Organe mit von der Tuberculose befallen sind.

Ich brauche hier nicht auf eine schwierigere Discussion einzugehen, in wie weit die epithelialen Elemente selber Bestandteile des Tuberkels bilden; ich weise nur auf die gröberen Thatsachen hin, die schon Virchow bekannt waren, dass z. B. bei der miliaren Tuberculose der Lungen fortwährend miliare, käsige Pneumonieen vorhanden sind, d. h. dass mit einer Infection der Lungen auf dem Blutwege sofort, wie wir uns heute nach Kenntnis der Bacillen überzeugen können, die Bacillen in die Alveolen hineingeraten und das Parenchym inficieren. Genau so verhält es sich in der Niere, bei der meine Untersuchungen schon vor 20 Jahren, unmittelbar nach der Entdeckung des Tuberkelbacillus, darauf hinwiesen, dass bei den mannigfachen Arten der Tuberculoseerkrankungen sofort die Harnkanälchen von dem Tuberkelbacillus befallen werden und in eine eigene Erkrankung verfallen, in der sich, bisweilen äusserst massenhaft, Bacillen befinden. Wir sehen dann in den Sammelröhren, in den graden und den gewundenen Rinden-Kanälchen eine Masse von Bacillen theils in Zellen, theils zu Cylindern zusammengeballt, die uns zeigen, dass die epithelialen Bestandteile der Nieren inficiert sind.

Diese Thatsache allein ist aber noch nicht genügend, um eine Verbreitung auf dem epithelialen Wege zu statuieren; denn wir sehen hier, dass zunächst die Natur sich selbst schützt, indem die kleinen Epithelkanäle verstopft werden. Die Lunge vermag dann nicht mehr zu respirieren, in Folge dessen wird aus den durch die Erkrankung befallenen Teilen der Bacillus nicht verbreitet.

Dagegen sehen wir, dass dieser Vorgang eintritt, sobald die Ausführungsgänge der Drüsen mit befallen werden. Diese Verhältnisse sind seit langer Zeit, schon vor der Zeit als der Tuberkelbacillus gefunden wurde, in der Lunge erkannt. Wir wissen von Virchow, dass die Erkrankung der kleinen Bronchien einer der wichtigsten Verbreitungswege und Ausgangspunkte der tuberculösen Erkrankungen der Lunge ist, und nach der Auffindung der Tuberkelbacillen können wir uns ebenso überzeugen, dass beim Fortschritt der Erkrankung in jeder Weise Tuberkelbacillen in die Bronchien hineingeraten. Es gehört bei der Lunge durch die complicierten Verhältnisse des

Digitized by Google

Organs nicht jedesmal eine eigentliche Erkrankung der Bronchien dazu, sondern die erweichenden käsigen Herde des Lungengewebes können in die Bronchien hineingelangen und sie inficieren; andrerseits wissen wir durch die Untersuchungen von Birch-Hirschfeld, dass eine eigentliche Erkrankung, eine käsige Entzündung der Bronchien den Ausgangspunkt für die Lungenerkrankungen giebt.

Wenn die Bacillen in die Ausführungsgänge der Drüsen gelangt sind, sind die Gelegenheiten für die Weiterbeförderung mannigfach. In der Niere sind es besonders die graden Harnkanälchen und die Sammelröhren, die als solche Stellen zu betrachten sind, von denen aus der Tuberkelbacillus eine weitere Verbreitung finden kann, während die tubuli contorti verstopft werden und keine weitere Verbreitung veranlassen. Daher haben die zahlreichen Tuberkel in der Rinde gewöhnlich keine Verbreitungstendenz, während die Tuberkel in der Marksubstanz sich vergrössern und die Ausgangsstelle der Phthisis renalis bilden.

In der Leber sind es die Gallengänge, in den männlichen Geschlechtsorganen sind es die Kanäle der Epididymis, die von der Tuberculose befallen werden und die Ausbreitung der Tuberkelbacillen vermitteln.

Wenn die Drüsengänge durch Tuberkelbacillen überschwemmt sind, dann wissen wir, dass letztere Gelegenheit haben, nach beiden Seiten sich auszubreiten, nach oben und nach unten. Am klarsten liegen die Verhältnisse in der Lunge, wo wir sehen können, dass sowohl der Exspirationsstrom wie der Inspirationsstrom im Stande ist, die Tuberkelbacillen nach beiden Seiten zu befördern und in die Gewebe zu verteilen; aber wir sehen in der Lunge, dass nicht überall, wo Tuberkelbacillen auf solche aprioristisch construierte Weise hingelangen, auch unmittelbar tuberculöse Eruptionen erfolgen. Wieviel Tuberkelbacillen muss ein Phthisiker durch seine Trachea und seinen Larynx befördern, ehe er eine Tracheal- und Larynxtuberculose bekommt. Das ist ja bekannt, und wir dürfen uns sagen, dass die Schleimhaut dieser Teile eine gewisse Widerstandsfähigkeit gegen die Tuberkelbacillen besitzt.

Sehr viel schwieriger ist es zu erklären, warum nicht, wenn einmal ein Bronchus inficiert ist, sofort die ganze Lunge von dem Virus inficiert wird; denn wir könnten uns vorstellen, dass, wenn die Tuberkelbacillen durch den Exspirationsstrom aus einem inficierten Bronchusgebiet herausgeschleudert werden, sie durch den Inspirationsstrom sofort in ein anderes Gebiet und dann über die Bifurcation hinaus selbst in die andere Lunge hineingelangen müssten. Und doch sehen wir, dass die Tuberculose in der Lunge lange stationär bleibt. Die Ursache dieses Verhaltens kann ich nicht auseinandersetzen, aber ich möchte auf eine Thatsache hinweisen, bei der wir diese Verhältnisse klar entwickelt sehen. Es giebt Fälle, die Ihnen alle bekannt sind, bei denen tuberculöse Bronchialdrüsen in einen der Hauptbronchien durchbrechen und von da aus das Tuberculöse Virus in den Bronchialbaum ergiessen. In diesen Fällen ist ausschliesslich dasjenige Bronchialgebiet von der Tuberculose befallen, in welches die Drüse eingebrochen ist. Wir sehen hier also, dass es zunächst lediglich der Inspirationsstrom ist, der von hier aus Tuberkelbacillen in die Lunge zu führen vermag, während die abfliessenden Bacillen nicht

Digitized by Google

sofort in andere Bronchien gebracht werden. Man ist im Stande, beiderartigen Kinderlungen—es ist namentlich im Gefolge von Kinderkrankheiten, wo diese Tuberculose auftritt—für den kleinen Teil der Lunge, den man tuberculös findet, durch Präparation des zuführenden Bronchus mit Sicherheit die Drüse aufzufinden, die in diesem Falle die Gelegenheit zur tuberculösen Erkrankung gegeben hat.

Sehr viel discutiert sind die Verhältnisse der Ausbreitung im Urogenitalsystem, und noch auf dem letzten Chirurgen-Congress, im April, 1901 sind sie der Gegenstand einer Ausführung unseres bekannten Pathologen von Baumgarten gewesen. Man hat gemeinhin angenommen, dass im Urogenitaltractus die Tuberculose nach allen Seiten sich ausbreiten kann. Diese Richtungen sind sehr mannigfach. Wir kennen schon, dass von einer Niere durch das Nierenbecken, der Ureter und die Blase abwärts inficiert wird. Wir nahmen bisher an, dass alsdann die Möglichkeit besteht, dass von der Blase aus aufsteigend durch den anderen Ureter und das andere Nierenbecken die andere Niere inficiert werden kann, ferner, dass von der Blase und der Prostata aus durch das vas deferens die Epididymis und die Testikel inficiert werden können. Ebenso galt als möglich, dass umgekehrt von einer Epididymis über vas deferens und Prostata der andere Hoden die Blase und die Nieren inficiert werden können.

Baumgarten hat nun die Behauptung aufgestellt, dass diese Möglichkeit nicht bestehe, dass vielmehr in Folge der Unbeweglichkeit des Tuberkelbacillus die Verbreitung nur auf dem Wege des Sekretstromes zur Blase und zur Prostata erfolgen kann. Er hat aber dabei offenbar die Thatsache übersehen, die physiologischexperimentell festgestellt ist, dass die *antiperistaltische* Bewegung es ermöglicht, dass das Secret auch von den abliegenden Kanälstrecken in die höher gelegenen gelangen kann. Diese antiperistaltischen Bewegungen sind im Ureter nachgewiesen, im vas deferens freilich noch nicht, aber wir können nach den Erfahrungen, die wir an jedem anderen musculösen Tubus machen, dass nämlich beim Verschluss des unteren Endes—und das ist die Vorbedingung—antiperistaltische Bewegungen eintreten, annehmen, dass auch im vas deferens eine antiperistaltische Bewegung stattfindet.

Dass die Epithelkanäle auch weiter entfernte Metastasen erzeugen können, wissen wir, da feststeht, dass der Darmkanal von der Lunge aus inficiert werden kann. Ich habe hier aber in Hinsicht auf die interessante und hochwichtige Erfahrung, die wir vorgestern von Herrn Koch haben vortragen hören, darauf hinzuweisen dass auch in diesem Falle von der Verbreitungsmöglichkeit durch den Larynx und Pharynx und den Digestionsapparat bei weitem nicht in Jedem Falle Gebrauch gemacht wird; wir müssen doch annehmen, dass Tuberkelbacillen aus der Lunge des Phthisikers unausgesetzt durch den Larynx, den Pharynx in den Darmkanal hineingelangen, ohne dass dadurch der Darmkanal sofort tuberculös inficiert zu werden braucht.

Ich verlasse das Gebiet der Epithelkanäle und wende mich zu den anderen Kanälen, welche die Organe besitzen, und die für die Ausbreitung der Tuberculose eine Wichtigkeit beanspruchen.

Wir kommen zunächst zum Lymphapparat. Dass die Lymphgefässe eine

Digitized by Google

wichtige Gelegenheit für die Ausbreitung der Tuberculose geben, ist auf der Hand liegend; ältere Erfahrungen, die schon vor der Zeit des Tuberkelbacillus gemacht sind, haben uns denjenigen Ort kennen gelernt, bei dem wir die Verbreitung innerhalb der Organe durch die Lymphgefässe am bestem studieren können, das ist der Darmkanal und die Chylusgefässe. Wir sehen hier, dass von einem tuberculösen Geschwür und durch die übrige Darmwand hindurch, die nicht von der Tuberculose zerstört zu werden braucht, mit Hilfe der Lymphgefässe, und des Chylusstroms der Tuberkelbacillus in die subserösen Gefässe und Mesenterialgefässe hineingelangen kann und die Lymphdrüsen inficiert. Dieser Weg wird nicht dadurch eröffnet, dass die Lymphgefässe selbst erkrankt sind. Wir werden darauf noch zurückzukommen haben; wir müssen die Erklärung Kochs acceptieren, dass es in erster Linie die Leukocyten sind, die in die Lymphgefässe mit den Tuberkelbacillen hineingeraten und in den Lymphdrüsen festgehalten werden.

Zu einer allgemeinen Infection des Körpers ist auf diesem Wege der Lymphbahnen eine Verbreitung der Tuberkelbacillen darum nicht möglich, weil nach den Untersuchungen Weigerts die Lymphdrüsen einen sicheren Abschluss ergeben, durch den die Tuberkelbacillen nicht weitergelangen können. Nur wenn, wie Baumgarten nachgewiesen hat, die Lymphdrüse selbst erkrankt, wird dadurch eine nächste höhergelegene Lymphbahn inficiert. So können allerdings schliesslich die Bacillen in den Ductus thoracicus und von da aus in den Blutkreis hineingeraten. Dass in diesem Falle die tuberculöse Erkrankung der Lymphgefässwand selbst als Eingangsstelle für die Tuberculose beim Lymphapparat unwesentlich ist, ist klar. Wenn wir die Verhältnisse mikroskopisch studieren, können wir uns überzeugen, dass die Lymphgefässe der Organe bei der tuberculösen Erkrankung vollständig obliterieren, und also auch der Lymphstrom in ihnen aufhört.

Ein grosses Interesse für die Verbreitung der Tuberculose im Organismus hat das Blutgefässsystem in Anspruch genommen. Wir können uns überzeugen, dass die Blutgefässe bei jeder tuberculösen Erkrankung miterkrankt sind, dass eine Blutgefässtuberculose bei jeder Erkrankung irgend eines Apparates miteinherläuft. Die neueren Methoden der Darstellung der elastischen Fasern und damit der Untersuchung der Wand der Blutgefässe haben es ermöglicht, selbst die kleinsten Blutgefässe in den Organen zu studieren und auf eine Beteiligung bei dem tuberculösen Process zu untersuchen. Es ist auf diese Weise besonders vor einigen Jahren Rieder gelungen, zu zeigen, dass bei jeder tuberculösen Erkrankung die kleinen Blutgefässe mitbeteiligt sind. Bei den Arterien war es bekannt; bei den Venen, die wir sonst schwer in den erkrankten Organen verfolgen können, sind auch auf diese Weise besondere Erkrankungen nachzuweisen.

Eine solche Erkrankung der kleinen Blutgefässe ist aber noch nicht Gelegenheit für eine Gesamtinfection des Organismus, weil sie beim Fortschreiten des tuberculösen Processes vollständig obliterieren. Wir wissen, dass sowohl Arterien wie Venen, wenn sie vom tuberculösen Process befallen sind, vollständig obliterieren und keine Gelegenheit zur Verbreitung der Bacillen geben. Es ist das die Widerlegung einer Auffassung, die Ribbert neuerdings

Digitized by Google

ausgesprochen hat, dass die Erkrankung der kleinen Blutgefässe für die Verschleppung der Tuberkelbacillen in den Organen eine wesentliche Bedeutung besitze. Diese Erkrankung der kleinen Blutgefässe zeichnet sich durch Bacillenarmut aus und zweitens sind die obliterierten Gefässe nicht im Stande die Bacillen weiter zu befördern.

Die grosse Bedeutung der Blutgefässerkrankung macht sich erst geltend, wenn Blutgefässe von einer gewissen Grösse hineingezogen werden, Gefässe, die nicht unmittelbar beim Eintritt der Tuberculose vollständig obliterieren, und bei denen die Möglichkeit nicht ausgeschlossen ist, dass Tuberkelbacillen in das strömende Blut hineingelangen. Dieser Vorgang, der für den Ductus thoracicus, für die grossen Arterien und Venen eine grosse Bedeutung hat, ist zuerst in dieser Stadt von Astley Cooper studiert worden und wir haben Gelegenheit, in unserem Museum die Präparate Cooper's zu sehen, die die Grundlage für unsere jetzige Kenntniss der Blutgefässtuberculose bilden. (Applause.)

In neuerer Zeit sind die Untersuchungen fortgeführt worden durch meine Landsleute Karl Weigert, durch Ponfick; für das Herz haben eine grössere Anzahl französischer Autoren, wie Letulle, Léon Petit und Hanot, sich der Sache angenommen, und, wie ich aus unserem Museum ersehen habe, haben auch hier Forscher diese Frage weiter in Angriff genommen.

Diese Erkrankung der grossen Blutgefässe, die in der That die einzige vollständig ausreichende Erklärung für die Verbreitung der Tuberkelbacillen durch den ganzen Körper giebt, ist zuerst von Weigert auf einen Durchbruch von tuberculösen Herden in die Blutgefässwand zurückgeführt worden. Ich habe gezeigt, dass dieser Weg nicht der einzige ist. Ausser dieser Möglichkeit ist noch die Möglichkeit vorhanden, dass durch Auftreten von tuberculösen Metastasen in der Intima der Blutgefässe eine Infection der Gefässwand erfolgt; dieser Vorgang der Intimatuberculose tritt uns ebenfalls in mehreren Varianten entgegen. So können erstens die Metastasen in einer ganz normalen Wand auftreten; sie können ferner als secundäre Infection bei einer durch andere Erkrankungen zerstörten Gefässwand sich zeigen. So habe ich an einer atheromatösen Aorta tuberculöse Thromben gefunden, oder neben verrucöser Endocarditis auf den Herzklappen Tuberculose constatiert. Endlich ist die Möglichkeit vorhanden, dass Thromben, die von einer Weigertschen Durchbruchstelle fortgeleitet werden, in anderen Blutgefässen die characteristiche Gestalt der tuberculösen Endangitis annehmen.

Beide Arten der Gefässtuberculose, die Weigertsche wie die primäre Erkrankung der Gefässintima, sind am Herzen, an Venen, Arterien und dem Ductus thoracicus beobachtet worden. Es wäre falsch anzunehmen, dass in jedem Falle, wo ein Tuberkel der Gefässwand sich entwickelt, gleich auch kolossale Mengen von Bacillen in den Blutkreislauf eingeschwemmt werden müssen. Von dieser Möglichkeit macht auch hier nicht der Organismus in jedem Falle Gebrauch und er ist auch vielfach durch Schutzvorrichtungen gegen diese Ereignisse geschützt. Oft wird der Gefäss-Tuberkel durch Fibringerinnsel abgedeckt, er kann durch Thrombose vollständig abgeschlossen werden. Ich habe oben ein Präparat eines tuberculösen Ductus thoracicus

Digitized by Google

aufgestellt, wo ein grosser Teil desselben durch einen vollständig organisierten und kanalisierten Thrombus verschlossen ist, so dass wir sicher sein können, dass zeitweise kein Tuberkelbacillus aus der tuberculösen Erkrankung des Gefässes in den Organismus gelangt ist. In einem anderen Falle kann ich Ihnen zeigen, dass in den Wänden der Lungenvenen eines Kindes an einer grossen Anzahl von Stellen sich Tuberkel entwickelt haben, von denen einige durch Fibringerinnsel abgedeckt sind, andere durch Organisation unschädlich gemacht sind, eine Anzahl aber durch Ulceration zerstört worden sind. Selbst aber, wenn dies eingetreten ist, so kann der Gefässtuberkel sich verschieden verhalten. Solche Tuberkel, die sehr arm an Bacillen sind, werden nur wenig verstreuen können; wir haben aber auch solche, von denen ich einige aufstellen werde, namentlich Herzklappentuberkel, in denen ganz enorme Mengen von Tuberkelbacillen an der Oberfläche der Gefässwände liegen. Wenn diese Mengen von Bacillen ins Blut gelangen, ist Gelegenheit für eine Ueberschwemmung des Blutes mit Bacillen vorhanden, und wir sehen das Auftreten einer ausserordentlich grossen Zahl von Tuberkeln: die Entstehung der akuten Miliartuberculose.

Es ist ferner hinzuweisen, dass auch in den Fällen von Gefässtuberculose wo nicht eine solche Menge von Tuberkeln entsteht, und also nicht Tuberkelbacillen selbst massenhaft ins Blut hineingelangt sind, doch Toxine in das Blut diffundiren und dadurch die Erklärung geben für das Fieber, das beim Bestehen von Gefäss Tuberkeln einen wesentlichen Bestandtheil des klinischen Bildes der akuten Miliartuberculose bedingt.

Ob diese Verstreuung durch direkten Einbruch die einzig mögliche Erklärung für die akute Miliartuberculose ist, oder ob noch eine andere Hypothese zu Hilfe genommen werden muss, hat in der letzten Zeit die deutschen Forscher viel beschäftigt und ihnen zu erregten Discussionen Anlass gegeben. Ich weise darauf hin, dass Ribbert, der zuerst angenommen hatte, dass die Tuberkelbacillen im Blute sich vermehrten, seine Behauptung Schritt für Schritt zurückgezogen hat und jetzt die Behauptung aufstellt, das in den sekundären metastatischen Organtuberkeln neue Infectionen der Blutgefässe durch kleine Gefässtuberkel entstehen. Diese Möglichkeit ist nicht zu leugnen, aber sie giebt erst eine Begründung für die Verbreitung der Tuberkelbacillen, wenn die Tuberkelbacillen in diesem Falle nachgewiesen werden und wenn die Ulcerationen der Tuberkel gefunden werden. Ich habe in meinen Conclusionen darauf hingewiesen, dass wir in den Beobachtungen Ribbert's vielleicht eine Erklärung für die von Weigert und mir beobachtete progressive Miliartuberculose finden, wie sie Versuchstiere und Kinder vielfach zeigen.

Ich verlasse das Gebiet, da meine Zeit abgelaufen ist, und weise nur noch darauf hin, dass ich die Ehre haben werde, im Museum Ihnen eine Anzahl mikroskopischer Präparate zu zeigen, und zwar in drei Serien, die die lokalen Veränderungen durch die Tuberkelbacillen, die käsige Pneumonie und die Veränderungen am Ductus thoracicus und an den Harnkanälchen zeigen. Ich werde in einiger Zeit die Präparate wechseln und bitte sie in Augenschein zu nehmen.

Ich hoffe als Grundlage dieser Discussion die Behauptung aufstellen zu

Digitized by Google

können, dass wir in den Verbreitungswegen der Tuberkelbacillen im Organismus eine wichtige Begründung der Variationen, in denen die menschliche Tuberculose im Organismus auftritt, erblicken können. (Applause.)

The PRESIDENT: Professor Benda has specimens upstairs, and has offered to demonstrate them. I think it would scarcely be wise to examine those specimens now. We have had a very good description of what we may expect to see, but I have no doubt there are some who take a special interest in the subject, and who would like to see them and hear the demonstration; they will have an opportunity at a later stage. I will now, therefore, ask Professor Hamilton to second—if I may say so—what Professor Benda has told us in opening the discussion.

Professor D. J. HAMILTON, *Aberdeen*: "On the Interpretation of the Lesions Caused by the Bacillus Tuberculosis."

Introductory.—Since the time of Laennec and that of Virchow's early publications, the tissue manifestations of tubercular disease have been matter of such close observation that it might be imagined we know all that can be known of the subject. Even yet, however, there remains something to be done; for we know not, for instance, why the bacillus selects certain tissues to grow upon and why others remain almost immune; why in some instances it remains localised in a particular focus, such as a joint, a limited set of glands, and so forth, while in others it spreads diffusely, and the portal by which it gains entrance to the body, in tuberculosis of deep-lying parts, is in many cases hard to perceive. All these matters are of primary importance in the study of the malady, but as yet they are wrapt in comparative mystery.

Bacillus sometimes fails to stain.—The presence of the tubercle bacillus in the affected part, and more particularly its inoculability upon a fresh host, are the great criteria by which we are enabled to pronounce upon the tubercular nature of a given disease. The latter is by far the surer of the two procedures, for while in many of the morbid growths which, from their structural features and degenerative tendencies, we would certainly pronounce to be tubercular, such as some miliary eruptions in the lung, lupus, and tubercular disease of bone, it is sometimes difficult to demonstrate the presence of the bacillus with the microscope, they nevertheless respond to the inoculation test. It has been alleged by Mr. Watson-Cheyne and others that in bone lesions, at least, the bacillus passes into a state in which it fails to elicit the staining reaction, and yet apparently is in a condition quite active enough to induce a tubercular eruption when transferred to the tissues of a suitable host. I often think that in old caseous parts, more particularly in the ox, it assumes this character.

Portals by which the bacillus enters the body.—The chief portals by which the organism gains entrance to the body are undoubtedly the lung and the lower half of the small intestine. In man, the upper air-passages and higher segments of the alimentary tract seem to be seldom affected primarily, while the lung itself, and to a much less extent the ileum, are the readiest points of

Digitized by Google

natural inoculation. I have met with only one instance of tuberculosis originating in the mucosa of the human stomach, while it is certainly rare to find the duodenum and upper two-thirds of the jejunum the seat of the disease. The pharynx and œsophagus, likewise—in man at least—are only rarely primarily tubercular. There is something wanting, apparently, in the natural conditions of the parts composing the upper portion of the alimentary canal, to encourage the organism to take hold and fructify upon them. It cannot be the absence of absorbents in the mucosa, for these prevail along the whole tract. No doubt they are more abundant in the lower part of the small intestine than elsewhere, but this is no reason why, in this particular region, they should become tubercular almost to the exclusion of the same kind of absorbents higher up. This property of the absorbents of the ileum is not confined to the case of tubercle. The same tendency to absorb bacteria occurs in typhoid, and where septic matter is swallowed, say, from an abscess of the tonsil or soft palate, although in these diseases the property is not so exclusively confined to this part of the bowel as in the case of tuberculosis. It has been alleged that the acid reaction of the stomach has something to do with its failure to become tubercular, but with what show of reason it is difficult to decide. The upper respiratory passages, the lung, trachea, and large bronchi are also seldom the seat of what might be regarded as the portal of entrance, while the vesicular tissue of the lung more frequently contains the primary nidus than any other tissue in the body.

The lymphatic glands along the course of the upper air passages, or those closely adjacent to them, as well as those in the neighbourhood of the upper segments of the alimentary canal, are nevertheless extremely common seats of a localised tuberculosis. This opens up the question as to whether the absorbents of mucous membranes can seize upon the bacillus and hand it on to the corresponding lymphatics without themselves becoming tubercular. My own impression is that they can; that the actual point of invasion may, from local conditions, prove unfavourable to the development of tubercle, and that the bacillus is passed on through them to a tissue more favourable to its growth.

The condition of the gum and jaw incidental to the presence of decaying teeth has been regarded by some as furnishing a point of access to the cervical glands, and I am myself constrained to believe that this may prove oftener an element of danger than is generally supposed. Many cases of tubercular enlargement of these cervical glands have come under my notice where, so far as could be ascertained, this was the only point of infection. But here, again, the bacillus does not apparently render the part on which it alights tubercular, but, being absorbed at the broken surface, is transmitted to a more vulnerable tissue. If anthrax gain entrance to the blood of the ox without generating a local lesion, if plague can infect the lymph glands through the skin, there is the possibility, at least, of something of the same kind happening with tubercle. How are we to account otherwise for localised tubercular foci in bone, and in the capsules and synovial membranes of joints, or for those cases of peritoneal tuberculosis without any evident lesion of the mucosa, but where the tubercles

Digitized by Google

notwithstanding are seen running continuously along the peritoneal lymphatics from the intestine upwards?

Then I expect the tonsil and the retro-nasal lymphadenoid tissue is a common seat of inoculation. The fact that they swell up so often in tubercular children shows that they are evidently stimulated by something of the organismal type, and I suspect that in a good many of these cases the bacillus of tubercle is the stimulating agent.

Diffusion of the bacillus.—Having gained access to the body, how does the bacillus spread abroad? There is good evidence to show that it tends to propagate along the lymphatics rather than along the blood-vessels. Indeed, I think it extremely likely that the bacilli of tubercle, taken directly into the circulation in small numbers, may be effectually disposed of by the blood itself. McFadyean's experiments seem to show that there is no direct relationship between the quantity of bacillus injected into the blood and the number of resulting tubercles. Sometimes the number is large and sometimes small with the same dose, evidently showing that many of the bacilli may be destroyed in circulating through the body. The organs are affected, moreover, in such experiments in by no means a regular order; the lymph-glands often escape or are only partially affected. No doubt when the bacillus gets into the blood circulation in large quantity, it induces in many cases a generalised eruption in which the lung participates most largely, but everyone will confess that in many cases of advanced primary tubercular disease of the lung, for instance, where it is hardly possible to believe that small numbers of tubercle bacilli are not absorbed by the blood-vessels from time to time, the other organs of the body may remain remarkably free from secondary tubercles. The organ which suffers most at second-hand from chronic primary tubercular disease of the lung is the intestine, and here the lesion commences in the absorbents of the mucosa, that is to say, in connection with its lymphatic system. The bacillus, which has been expectorated from the lung and subsequently swallowed, is appropriated by the intestinal absorbents, while little, if any of it, is conveyed to the intestine embolically by the blood-vessels. Illustration of the preference the bacillus displays for the lymphatic system is afforded time after time in localised tuberculosis of the lung. Say that a patch of cheesy tubercular pneumonia be present in the organ, nothing is more common than to see the lymphatics radiating from this infected with secondary tubercles, the lymphatics of the pleura similarly tubercular, and the large bronchial glands beset with grey nodules. Presumably the lymph-stream from the air-vesicles tends to proceed directly or indirectly towards the pleura and culminates in the bronchial glands. The bacillus is carried along with the stream and infects progressively the lymph-vessels in which it is contained. There has grown up a current belief, however, of late, that a lymphatic tuberculosis may be propagated along the lymph-paths in a direction opposed to that of the lymph-stream, and certain circumstances seem to lend support to the view. Renal phthisis may originate apparently from a tuberculosis of the lower genito-urinary organs by the bacillus making its way up the ureter, that is to say, in a direction opposed to the flow of urine along them just

Digitized by Google

as in septic disease; and, if this be the case, then there is every likelihood that tubercle may also be propagated along the lymph-paths in a direction opposed to that of the lymph-stream.

I have often been struck with the fact that in primary tubercular disease of the intestine and peritoneum the lymphatics of the capsule of the liver are studded with tubercles all along their course, while the liver substance remains free from their presence. This I can explain only on the supposition that the lymphatics are much more readily infected by the bacillus than the blood-vessels; that for some reason the bacillus disappears in the blood-vessels, while it continues to exist and to propagate in the lymph vessels. The immunity of the liver substance in such cases of abdominal tuberculosis is all the more remarkable when it is remembered that the hepatic capillaries tend to arrest particulate foreign matters passing along their channels. Quite possibly, if arrested by the endothelium of the capillaries, the endothelial cells dispose of tubercle bacilli as ordinary phagocytes do. Indeed, the probable reason for the preference shown by the tubercle bacillus for the lymphatics, as compared with the blood-vessels, may be that the lymphocytes contained in the lymph are not so phagocytal as the white cells of the blood. The fact that lymph-glands are so commonly a seat of tubercle shows that the cells within them are in nowise inimical to the growth of the bacillus.

Giant-cells.—Attention may be called here to the giant-cells of tubercle and their meaning. They are a constant feature of interstitial tubercles, but fail completely in primary tubercular pneumonia. That is to say, if the tubercular manifestation be in a connective tissue, there you will find giant-cells make their appearance as soon as the nodule becomes a naked-eye object, while in the case of pure and uncomplicated caseous tubercular pneumonia, there may not be found a single giant-cell in any part of an entire lung. While, should the pneumonia have passed the caseous or second stage and entered upon the third, or that of ulcerative softening, and should lymphatic infection of the organ have occurred as a result of the absorption of the bacillus from the ulcerating foci, these giant-cells become abundant in the secondary lymphatic tubercles. It is a remarkable circumstance that in some of the lower animals these giant-cells become packed with tubercle bacilli, while in man it seldom occurs that a single bacillus is found in their substance; in the one case their protoplasm is eminently phagocytal, in the other it, for some reason, does not possess this function. They are evidently overgrown cells of the connective tissue type, and for this reason are not encountered in pure tubercular catarrh. They evidently destroy the bacilli in certain animals while in others they appear to play a passive *rôle*, while, as the virulence of the tubercle poison wears itself out, they are resolved into fibrous tissue and disappear, just as in the case of the giant-cells of a myeloid sarcoma when the tumour takes on bone-forming tendencies.

Cause of localised and diffuse tuberculosis.—It seems to me, therefore, that the localised character of certain tubercular lesions must be accounted for by the destructive influence exercised by the blood upon the bacillus, accompanied by the fact that the number of bacilli absorbed is small. Quite

Digitized by Google

possibly the barrier of young living cells seen round chronic tubercles arrests a good many of the bacilli which otherwise might get into the blood, but one can hardly conceive that where the bacilli are fairly abundant some of them do not get absorbed by the blood-vessels. These, however, being in small quantity, are capable of being dealt with by the protective phagocytes of the blood. The cases most favourable to the calling forth of a general acute miliary tuberculosis are those where a tubercular focus begins to suppurate, where the cheesy dead tissue softens, where, presumably, the barrier of phagocytes at the margin has degenerated, and where the bacillus has increased to such an extent that the softened centre of the tubercular focus may often contain what closely resembles a pure culture of the organism. Under these circumstances the bacillus is rapidly absorbed by the blood-vessels and in such quantity that the natural scavenger-like properties of the blood leucocytes fail to cope with the excess of parasite, and an extensive crop of miliary tubercle is the result.

Immunity of certain tissues.—It is evident that, for some reason, the tubercle bacillus takes hold upon some tissues more than others. Thus the lung, intestine, spleen, kidney, liver, testis, the meninges, the pleura, the peritoneum, and lymph-glands of the trunk are all highly susceptible, while the muscles and the peripheral lymph-glands—in man at least—are seldom the seat of tubercular deposits. But of all tissues which enjoy this immunity perhaps the pericardium is the most remarkable. Tubercle of the pericardium is a very rare disease. I have seen only a few examples of it. The lung may be beset with tubercle; its pleura may be bound by inseparable adhesions to the pericardium and be itself extensively tubercular; the neighbouring mediastinal glands may be all tubercular, all the favourite seats of tubercle in other parts of the body may show the disease, and yet the surface of the pericardium remain quite smooth, glistening and unaltered. This applies both to the pericardium proper and to the epicardium, and the reason for it I fail to perceive. So far as its blood supply is concerned, there is no assignable reason, and its lymphatics have extensive collateral communications. Indeed, it seems just the sort of membrane to fall a prey to the ravages of the parasite of tubercle, and yet it almost always escapes. Is it that the pericardial serum is detrimental to the existence of the bacillus, and that when the membrane does become tubercular this property has been lost?

The winnowing action possessed by certain organs, such as the spleen, bone-marrow, and probably the vascular fringes of joints upon the blood corpuscles, and by the lymph-glands upon the lymph-cells, should predispose, it might be thought, to the entanglement of the bacillus and to the occurrence of tubercle within these organs, and quite likely that is so. The spleen and bone medulla are common seats of tubercle, and the bones possessing red marrow, such as those of the carpus and tarsus, the bodies of the vertebræ, and so on, are said to manifest this winnowing function in a pre-eminent degree, while they also show the greatest tendency to become tubercular.

Outward aspect of tubercular lesions.—The outward aspect displayed by the lesions generated by the action of the bacillus of tubercle varies perhaps more

Digitized by Google

than that of any other form of tumour disease. I met with a case lately, for instance, where the head of the pancreas, the omentum, and neighbouring viscera were bound up by adhesions into a tumour-like mass, having so strong a resemblance to abdominal cancer that it was only by microscopic examination and the identification of the bacillus that the true nature of the disease was discovered. In the horse it sometimes takes the form of a huge sarcoma-like growth, with little tendency to caseate, but absolutely beset with the specific organism; while in the cow, guinea-pig, and rabbit the inclination to caseate is extreme.

It may be said that in organs possessed of excretory ducts, and which are liable to tuberculosis, the disease may assume one of two garbs—either that of a miliary eruption in the interstitial tissue, or that of a tubercular catarrh of the ducts. The determination of the one or the other form depends apparently on how the bacillus gets into the organ. If carried in with the blood, the tendency is towards a primary eruption of interstitial tubercles, but, if the bacillus gets into the ducts it stimulates the epithelium primarily and induces a catarrh which terminates in caseation and ulcerative softening. In tubercular pneumonia and in ordinary renal phthisis the disease appears to be of the latter variety, and a natural question to ask in the case of renal phthisis is how the bacillus gets into the urinous tubes. There are two possibilities, namely, that it is excreted from the blood along with the urine, and renders the tubes tubercular in its passage downwards, or that it makes its way upwards from the lower genito-urinary viae just as other organisms undoubtedly do. In support of the former view it has been said that tubercle bacilli have been found in the urine where there was an entire absence of tubercle in the genito-urinary system.

Origin of tubercular pneumonia.—In regard to tubercular pneumonia, it may be asked whether it is always caused by the bacillus being inhaled. Most cases seem to start in this way—but several cases which have come under my notice lend support to the view that a typical caseous pneumonia may follow auto-infection from a distant focus. Thus I have seen a primary abdominal tuberculosis induce an eruption of nodules in the lung which were eminently catarrhal-pneumonic, in which the disease was intra-vesicular, and in which there was a complete absence of giant-cells.

Caseation.—The extraordinary regularity with which caseation takes place in tubercle of all kinds is one of its most notable features. Almost as soon as it becomes an object visible to the naked eye this necrosis commences to show itself, and it is this necrosis which encourages ulceration in an organ infiltrated with tubercle. Various theories of the cause of this tissue-death have been held from time to time, but the one which meets with most favour is that there is secreted by the bacillus a substance which has a caustic influence upon the surroundings and which destroys them. This substance is probably contained in the bodies of the parasite. Tuberculin does not induce the necrosis, while dead cultures, when injected, in addition to occasioning an eruption of tubercles closely resembling those set up by the living bacillus, have a certain tendency to cause this peculiar variety of tissue-death. The substance,

Digitized by Google

whatever it be, must be extremely powerful, for often in young tubercles, where the bacillus can hardly be detected, the caseation is far advanced.

Absence of putrefactive odour.—The absence of putrefactive odour in ulcerating tubercular disease is another remarkable feature which has never been thoroughly explained. Artault and others have shown that phthisical cavities in the lung contain numerous bacilli and cocci other than the bacillus of tubercle, and yet it is a remarkable fact that the odour of such phthisical cavities, although peculiar, is not by any means that, for instance, of a gangrene, and the breath of the individual need not possess any odour of putrefaction.

The PRESIDENT: We have listened with very great interest to the two papers by which the discussion has been opened, and we have the basis here, I think, for an exceedingly interesting debate. We appear to have the men amongst us to take part in such a discussion. It appears to me that there are one or two special points on which Professor Benda and Professor Hamilton agree entirely, and I think during the discussion we shall get some additional evidence in favour of those points. I think perhaps the most interesting at the present stage is the conclusion which appears to have been arrived at by these gentlemen, that it is not necessary for the invasion of the tubercle bacillus that there should be a local lesion. That appears at present to be an exceedingly important question.

As to the spread of the tubercle bacillus when it gets into the body, there can be little doubt that there may be a considerable amount of difference of opinion. However, I think we have here the basis on which to found an admirable discussion, and I shall first ask those gentlemen whose names appear on the printed list to speak, and then those who have also consented to join in the discussion. In the meantime, I should be glad if other gentlemen who are here would send up their names. I do not want to call upon a man if he is not willing to speak, but I see before me a number of gentlemen who are well qualified to send up their names. I will call upon Dr. Habershon.

Dr. S. H. HABERSHON, *London*: Mr. President and Gentlemen,—I think we are greatly indebted to Professor Benda and Professor Hamilton for their contributions to this interesting discussion. I will only make a few remarks as representing that enormous field for tubercular pathology which we have at the Brompton Hospital in London, as one of the physicians who held the post of pathologist there for about eight years. During that time we did nearly 2000 post-mortems on tubercular cases alone, and I should like to add what is our strong belief there on the several points which have been indicated. I think the forms of tuberculosis and its manifestations in the body depend, first, upon the mode of its entrance; second, upon the character of the invasion; and, thirdly, upon the resisting power of the individual. On this last head we have not heard very much.

I shall concentrate my attention on a few of the facts which I think some

Digitized by Google

of our post-mortems show as to the entrance of the tubercle bacillus into the body. These facts are confirmed largely by some interesting experiments conducted by Dr. Sidney Martin when engaged in the work of the Tuberculosis Commission in London. In some large feeding experiments on lower and less resistant animals, in which food was given containing human tubercular sputum and other matter containing the tubercle bacillus, he found the modes of invasion were twofold—(1) through the tonsils; (2) through the lower part of the small intestine. Professor Woodhead is much better acquainted with the work of this Commission and with Dr. Sidney Martin's work than I am, therefore he will no doubt correct me if I am wrong and misquote. Dr. Martin says he found the commonest modes of invasion were through the tonsils and through the small intestine. Sometimes the tonsil itself ulcerated, though that was rare. More often the cervical glands in the neighbourhood were infected without the tonsil itself being invaded. In other cases the intestines were clearly the point of entrance; and in those two manifestations were seen. Sometimes one or two small recent tubercular ulcers were seen with tubercles in the lymphatics outside. In others glandular tuberculosis was present in the mesenteric glands, but nothing was seen in the shape of an ulcer of the intestines. These experiments seem incontestable, and agree with the views already expressed by the two distinguished men who have already spoken to us.

In Brompton Hospital, of course, we always look for our material in acute cases to aid us in the matter of determining the path of entrance. In the more chronic cases there are so many organs affected that the original path of entrance very often seems to be lost. But I think we may say most certainly that we do have indications in the acute cases of entrance through an epithelial structure by some channel, either the small bronchial glands, or through the genito-urinary tract, or through the intestines. I think we sometimes have distinct evidence that invasion can take place through a membrane without affecting the membrane itself. In support of that I will quote one or two cases. A young girl, whose illness I had the opportunity of watching from the beginning, was taken ill with an insidious attack of pleurisy, ending in effusion. I say "insidious" because it was not an acute disease at first; the temperature was very low, not exceeding 100° or 101° F., with scarcely any signs of general illness. This patient developed pleural effusion, and I took her into the Brompton Hospital, where she remained two or three months, and eventually died. Post-mortem we found acute tubercular pleurisy, without invasion of any other organ in the body, except some large tracheal and tracheo-bronchial glands. There was no invasion of the lung, except one or two tiny nodules, which had evidently spread from the pleura. This was thick and cheesy, and the deposit appeared on the visceral and costal surfaces of the pleura; she died of exhaustion. How did that tubercle affect her? Here was one of those rare cases, which anyone can see in the hospital museum, where there was no primary invasion of the lung. The only glands affected were those in the trachea, and the only acute lesion was that of the costo-visceral pleura.

Digitized by Google

Another group of cases in which we have ventured to think we could find the source of invasion was in some of the cases of acute generalised tuberculosis. In some of them the only lesions—and Dr. Martin emphatically agreed with me in this—which could be discovered that could be supposed to be the sites of entrance were one or two small ulcers in the intestine. These seemed to point to tubercular food as the source of the trouble. But there was no chronic lesion; there was acute caseous tuberculosis, a very large and abundant crop of mesenteric glands, acute caseous peritoneal tubercle, tubercle in almost every organ of the body. Even in the heart there were small yellow tubercles, on the septum between the ventricles. Indeed there was abundant miliary tuberculosis—large caseous nodules in the spleen, and abundant caseous nodules the size of a small pea throughout the whole lung. This was certainly an acute case, and from the immense number of mesenteric glands and the two or three ulcers in the intestine, we thought it was one of those cases in which the invasion was through the intestines; it had left its mark in the small recent ulcer.

Time will not permit me to go into other cases. The only other set of cases I might quote as having given us some guidance are those in which there was acute disease and in which the major part of the stress fell upon the genito-urinary tract. In one woman there was extensive ulceration of the lining membrane of the uterus, with excessive caseous disease in both Fallopian tubes, the ovaries were diseased, there was abundant peritoneal tubercle, and acute tuberculosis of other organs. So I think the evidence derived from a large number of cases at Brompton Hospital shows these two facts—that invasion can take place, probably through the tonsillar epithelium, without necessarily invading the tonsils themselves. And then I think we have evidence that the intestinal mucous membrane in the lower part is a very great source of invasion, sometimes leaving a trace of this invasion, sometimes giving no clue at all.

But we also, I think, have evidence that the urinary and genital organs are the sources of entrance in a few cases. I believe the different forms of acute disease show that the tubercle bacillus, with its resultant chemical activity, must vary very much in different cases. I think it is sometimes very difficult to tell whether the particular manifestation is due to the unequal resisting power of the host or to some peculiarity of the bacillus. I have formed the opinion—though perhaps it is only based upon rather loose observation—that the tubercle bacillus itself does vary very much in virulence and in the toxines which are produced by it. Otherwise I do not know why we have in one case—whether the spread be through the lymphatics or through the blood-vessels—acute miliary tuberculosis, leading, if the patient live sufficiently long, to acute necrosis of tissue; while in other cases we have an abundant manifestation of nothing but caseous nodules, without the smallest tendency to necrosis. No doubt, as Professor Hamilton has said, the recently accepted view seems the most probable one—that in these cases of caseous disease the peculiar toxine which produces necrosis of cells is present. But I think the tubercle bacillus itself varies very much,

Digitized by Google

apart from the resisting power of the host. In the latter case the resisting power of the host has a profound effect in determining whether the disease is acute or whether it will remain chronic. We have had a very definite group of cases at Brompton Hospital in a certain series of years in which syphilis was present in the patient. Syphilis produces a profound degenerative effect upon the body. In a large number of cases there was tubercle present, and in every instance there was gangrene. If I were asked what is the most potent cause of gangrene in tubercular cases, I should say syphilis. We have had a definite group of such cases, and in every one of these gangrene was produced. In one case gangrene was the chief disease; and it was only after cutting a large number of sections of some of the septa between gangrenous cavities that we found tubercle bacilli in any abundance. I read a description of one such case before the Pathological Society of London some years ago.

Our Brompton Hospital experience shows us both the modes of invasion in some cases of acute disease and in chronic disease. There can be no doubt that the spread, after it has once entered the body, is in four different ways. By far the commonest mode of spread is, as we have heard, through lymphatic absorption. In other cases the absorption is undoubtedly through the blood-vessels. I would instance in support of this the acute miliary pulmonary tuberculosis which frequently occurs after a severe attack of hæmoptysis. In the hospital, as confirmed by the post-mortem records, there are cases in which a patient has had a sharp attack of hæmoptysis, which has been followed immediately by an acute miliary outburst in the lungs, and the patient has died in consequence in a few days. That is an instance of the spread of a vast number of tubercle bacilli into the general circulatory system, and the consequent discovery of miliary tuberculosis on the post-mortem table. We have evidence, in certain organs, of direct extension, like an advancing army. There is a line of tubercle across the lungs, and it advances in a direct way, as we all know, extending to the pleura, and so on. I think that method may be included as lymphatic absorption, because no doubt the spread occurs through the small lymphatics.

There is only one very rare mode of spread, and that is directly along the epithelial channels. Of that we have a very beautiful case, and I am sorry it is not in the museum here. It is one of extensive ulceration in the bladder, while there is an enormous mass of caseating disease of the ureter and tuberculosis of the kidney, with multiple tubercular manifestations elsewhere.

There is another rare case in which, apparently, the disease has spread directly down the bronchi, for all the bronchi are extensively ulcerated and bronchiectatic; and around these ulcerated tubercular bronchi there is a manifestation of tubercle in the neighbourhood. I cannot venture to take up your time further; but I wished to state what I think our experience in the Brompton Hospital has been during the last few years. (Applause.)

Professor OSLER, *Baltimore, U.S.A.*: I would like, first of all, to congratulate the Section on having had such a masterly presentation as that

Digitized by Google

to which we have listened from Professor Benda. I do not know that I have heard, for many years, any more delightfully graphic and lucid presentment of this extremely abstruse and complicated question. I should like particularly to call the attention of the clinical physician to the work which has been done of late years, particularly by Professor Benda, on this question. It has been summarised so fully and thoroughly by Professor Benda quite recently in the 'Ergebnisse' that it is now fully available in that publication for the information of those of us who are not ourselves working directly at pathological questions. I do not think I have read anything which so clarified my ideas with reference to the distribution of tuberculosis as that masterly analysis given by Professor Benda in the publication I have named. (Applause.)

The point I wish to speak of particularly is with reference to the so-called "typhoid" form of acute miliary tuberculosis. I think the view that Professor Benda has taken is correct—that the clinical manifestations of that form are very largely due to a toxæmia; that it is not itself directly due to the mass of tubercles, but to the toxæmia of local origin. If I understand him aright, he says it is due to the enormous number of bacilli growing directly into tuberculous endarteritis that is the source of the toxæmia, and not to the growth of small miliary tubercles throughout the body. That is an exceedingly important observation, and affords the clue to what would otherwise be difficult of explanation—the clinical variations in the forms of miliary tuberculosis.

With reference to the methods of invasion, in the serous membrane tuberculosis to which Dr. Habershon has particularly referred, I would like to make one or two remarks. In the first place, I cannot agree with my friend Professor Hamilton that tubercular pericarditis is rare. I think it is by no means uncommon; at least that has been my experience. Tubercular pericarditis is very often overlooked—it is almost always overlooked clinically, and I have known it overlooked pathologically, because tuberculous pericarditis may be so completely masked in fibrin and hæmorrhage, and the serous tuberculosis may be the only manifestation of the disease in the body. I have, on several occasions at societies, seen a case shown as ordinary pericarditis which was in reality tubercular. I think in this instance it is almost invariably an invasion from a mediastinal caseous lymph-gland which is attached to the pericardium.

A very important question is as to how the invasion occurs in acute pleurisy. There can be little question that the large proportion of cases of acute pleurisy are tubercular; and yet in a very considerable number of those subjects there is found, at post-mortems, no lesion of the lung. But you do find, I think, in all instances—it was so, I believe, in the case mentioned by Dr. Habershon—lesions in the bronchial or the tracheal glands. I think the invasion in the cases of acute primary pleurisy is almost invariably from the lymph gland or from a small focus of tuberculosis just beneath the pleura. How often do we find at post-mortems, in persons who have no other tuberculous lesions in the body, just little caseous masses, isolated perhaps,

Digitized by Google

one or two beneath the pleura and in different portions of the lung. They may not be caseous; they may be calcified; and I think these are sometimes the causes of acute tuberculous pleurisy. More frequently, I think, there is invasion from the bronchial glands.

I think this discussion on Professor Benda's paper will be of the very greatest service. I think that, and all this other work which has been done lately, shows us how much there is yet to do. We thought some years ago that nearly everything was known with reference to tuberculosis, but there are problems still awaiting us for solution.

Professor E. J. McWEENEY, *Dublin*: The remarks which we have just heard from Professor Osler deprive me of some points which I wanted to make. The first thing I wanted to say was to congratulate Professor Benda on the extremely able exposition which he gave us of the latest views as regards the diffusion of the tubercular bacillus in the system. I have never heard a more able and succinct account of this important and difficult question. If I may select one point out of the many which he made, I would choose for notice the demonstration which he gave, not only by word but by picture also, of the outbreak of miliary tuberculosis on the intima of blood-vessels, It has long been recognised that one of the chief methods by which miliary tuberculosis comes to pass is by the inbreak of tubercular material by means of ulceration into the wall of a blood-vessel. But what Professor Benda and his fellow-workers have demonstrated with so much perfection is that, apart from the solution of continuity in the wall of the blood-vessel, you may have an outbreak of miliary tubercle on the blood-vessel wall. I have never myself observed these miliary tubercles on the intima of small arteries, but I must also confess I have never made any very close search for them. Having heard Professor Benda's paper to-day, I propose in future to make careful examination in all cases of miliary tuberculosis which come under my notice, with this object in view.

There was one point upon which I should like to ask Professor Benda a question, and that is with regard to the frequency of genito-urinary tuberculosis. We admit that genito-urinary tuberculosis of renal origin can readily be accounted for by the excretion of tubercle bacilli circulating in the blood into the tubules of the kidney. But what I confess I am not clear upon is this: What is the cause of these frequent cases of genito-urinary tuberculosis, especially in male subjects, which appear to originate at the other end of the genito-urinary tract, that is to say, in the lower part, chiefly in the epididymis? Surely no one who has performed a large number of post-mortems at a general hospital can have failed to be struck by the frequency of apparently primary caseous foci in the epididymis. What is the cause of this frequent localisation of tubercle bacillus in the epididymis? Conceivably it might be that, owing to some injury, it may be a *locus minoris resistentiæ* for tubercles. But I should like to know whether there is any other explanation of it.

Another point which occurs to me as one upon which further information

Digitized by Google

is desirable is as to whether any large number of tubercle bacilli can circulate in the blood without producing miliary tuberculosis. According to the results obtained by Professor Adami with regard to the milk of cows which seem at any rate not to have tuberculosis of the udder, and according to the admirable paper which we heard from Dr. Rabinowitsch on the subject, it would appear that tubercle bacilli can be excreted by the mammary gland of the cow without producing localised tuberculosis; but how can the bacilli reach the mammary gland in those cases unless they circulate in the blood? Yet, by the terms of the proposition, they are not the subjects of miliary tuberculosis, but only of general tuberculosis. Why is it that in some cases tubercle bacilli can circulate in pretty considerable numbers without producing miliary tuberculosis, while in other cases miliary tuberculosis appears to be the result? Is it a difference in the resistance of the individual, or is it a difference in the virulence of the organism?

Professor Hamilton remarked on the fewness of bacilli in giant cells in the human subject. No doubt they appear to be few. But still, in a few cases where I went into the matter carefully by the method of sections, I found that tubercle bacilli were demonstrable in giant cells much more frequently than one would imagine if one only examined haphazard one section. If one goes to work by cutting the giant cell from end to end, in sections not more than 5μ in thickness, it is possible to demonstrate bacilli in a much larger number of cases than is thought to be usual. My own view as regards the nature of giant cells and their occurrence is that they are an expression of chronicity of the process and fewness of bacilli. When the bacilli are numerous and the toxine is great in quantity, you may have foci of necrosis formed, without the process running on to the formation of giant cells. That is the view which has been suggested to me as the result of whatever observations I have been able to make on the subject. In this connection I may mention the fact that from a careful study which I made of a case of acute miliary tuberculosis of the liver, of intestinal origin, I am inclined to think that an epithelial cell of the liver may be converted into a giant cell directly. I made a very careful series of sections in this case. The tubercles were extremely small, indeed they were invisible to the naked eye. They represented the very earliest stage of tubercle formation with which I am acquainted, namely, the presence of a single epithelioid cell, a single large swollen cell, with a faintly staining nucleus, sometimes two or three lymphoid cells around it. I concluded at the time, as the result of this series of sections, that these epithelioid cells were derived sometimes directly from epithelial cells—that is to say that they did spring occasionally directly from the transformation of epithelial cells.

As regards the frequency of tubercular pericarditis, I agree with Professor Osler. Several cases which, in my own pathological work, I have put aside as sero-fibrinous pleurisy of what I call the ordinary description, I have found on examination to be tuberculous in character. And if one cuts through the adherent layers of visceral and parietal tumour with a sharp knife, and looks very carefully at the portion of the exudate which immediately overlies the muscular tissue of the heart, one will see little dots of yellow caseating

Digitized by Google

material which would escape notice unless they were looked at very closely indeed. When these pieces of tissue are subjected to microscopical examination, typical tubercular lesions can always be found. One interesting fact is—it is an isolated fact, and I do not know whether it is of general application—that the last case of tuberculous peritonitis I was enabled to diagnose during life by the examination of the fluid obtained by paracentesis pericardii. The fluid was sent down from the ward to my laboratory, a perfectly clear fluid, apparently devoid of sediment of any kind. After vigorous centrifuging I obtained a very small amount of sediment, and on staining this by Gramme's method no micro-organisms were discoverable in it. The sediment consisted of polynuclear leucocytes of the ordinary kind. I stained the companion film for the tubercle bacillus, and in the first field I found a polynuclear leucocyte containing six tubercle bacilli. They were on each side of the nucleus. Perhaps I have not a sufficiently large experience to speak with authority as to the frequency of occurrence of bacilli in polynuclear leucocytes, but I confess that this was the first occasion on which I had ever seen them occurring in a polynuclear leucocyte.

The only other point I wished to make was this—and it shall be expressed in one sentence—whether there is anything to lead us to suppose that in certain circumstances the tubercle bacillus may either lose its staining power or pass into some condition in which it is not demonstrable by our present methods. I have had occasion more than once to examine cases where there were most extensive tubercular lesions, and even on the most perfect examination I have failed to discover tubercle bacilli at all. These cases are inoculable, but bacilli are not demonstrable in them. It may be heresy—and I believe it is—to even ask such a question; but every man must judge of these questions from his own work. Unless a man thinks for himself there is no use in his doing scientific work at all. I suspect that the tubercle bacillus may pass into some condition in which it is not quite demonstrable.

Sir CHRISTOPHER NIXON, *Dublin:* Mr. President and Gentlemen,—I have only one word to say to the Section, and that is to congratulate you upon the extremely interesting papers which have been brought forward, and the very able way in which they have been discussed.

The one point I wish to comment on is with regard to the character of the tubercle bacillus, as to whether, under certain circumstances, this bacillus, in the course of its life-history, may not be of extremely virulent character, and so give us an explanation of the extraordinary virulence of cases of acute tuberculosis as contrasted with the more chronic forms of the disease. I take it you have to regard the differences between the forms of chronic tuberculosis and the acute forms as due either to diminished resisting power on the part of the individual or an increased virulence in the micro-organism. And it seems to me there is a very strong analogy between conditions that you find in acute tuberculosis and those which occur in endocarditis. You find a case of endocarditis—I speak of the ordinary subacute endocarditis—in which there are precisely the same kind of organisms as in

Digitized by Google

the malignant. Those cases progress favourably, without any very serious or feverish symptoms, but in a certain proportion suddenly there is a disturbance in which the typhoid form of endocarditis is set up. How are you to explain that? Are you to explain that virulent form as due to a diminished power of resistance on the part of the individual, or to some change in the micro-organism? That, I think, is a very important point to consider, because if you admit that there can be some change in the character of the micro-organism which will be likely to affect the individual, is it not possible in the case of bovine tuberculosis that you may have a form of tubercle bacillus which is incapable of producing the disease in man, but which, under certain developmental conditions, may be communicable to man? It might then set up affections of the mesenteric glands, quite apart from direct communication of the disease to the intestine itself. I think that is a point of importance for the Section to direct its attention to—whether we are to assume that the tubercle bacillus in its ordinary life-history maintains a uniform standard as regards its nature, or whether, under conditions which may change that nature, it may produce a very virulent condition of disease in the individual. Further, the Section might consider whether, in the case of bovine tuberculosis, in the great majority of instances, it does not produce the disease in man, until some such change favours the development of the disease. That is the only point which I thought it would be of interest to bring before the Section. I agree with what has fallen from Professor Osler, which corresponds with the practice of my own hospital—that tubercular pericarditis is not an uncommon form of the disease, and I think it has very frequently been overlooked.

Professor J. G. ADAMI, *Montreal:* Mr. President and Gentlemen,—I have this serious disadvantage, that I did not hear Professor Benda's most admirable address. Until last year I had exactly the same idea that Dr. Hamilton has expressed, that tubercular pericarditis was rare, and I used to tell my students so. But we happened to run out of our old stock of material showing inflammation, and we had some new material, which we put aside—new cases, moderately severe, typical, as we thought, of serous or sero-fibrous pericarditis, subacute. In one we found tubercles and not in the other, and I had a difficulty in finding among my material any stage of pericarditis which was not tubercular. It certainly gave me a totally different idea about the frequency of it, which I had doubted before. While I am still somewhat doubtful, I confess that tubercular pericarditis is to be met with simulating distinctly ordinary simple subacute pericarditis.

Another point which has been brought forward this morning is that dealing with epithelial and tonsillar infection. It seems to me we must acknowledge freely—and the wonder is that after the many papers and researches which have been published during the last ten years on the tonsils and glands of the intestine we have not settled the point before—that the passage of bacteria into the system may take place through the epithelia. The more one studies the subject and the more one looks for it the clearer becomes the evidence

Digitized by Google

that bacteria are constantly passing through the tonsil and the intestine, and are as constantly being destroyed. The most remarkable of all these observations as showing the passage of particulate matter from the intestines are those which may be well known to many of you. I refer to the work of Mr. McCallum, my Toronto colleague in Canada. He fed the Lake Lizard with iron. Now, the Lake Lizard is an animal which you can keep fasting for eight months. He kept some of these without food for months, gave them their dose of iron, and killed them an hour or so later. On taking the whole of the intestines out and cutting sections of the small intestine and staining for iron, he saw every evidence of leucocytes in large numbers in the lumen of the intestine containing these blood granules of iron. He found them also in between the epithelial cells, and also in the blood-vessels of the liver and spleen. In a comparatively short time there had been a large passage into and out of the intestine. One can get similar results, but not so clear, if one examines the mesentery of perfectly healthy rabbits; and one can see that bacteria do pass in an apparently healthy animal where there is no sign of lesion in the intestine. The barrier against our accepting this is the difficulty in realising that it requires a large amount of bacteria to set up infection or a marked susceptibility, and the failure also to recognise that endothelial cells of certain organs, such as the liver, are constantly destroying bacteria, or have the capacity to do so. One has only to repeat Virchow's and other observations, and to inoculate bacteria into the general blood stream, and cut out the liver, and, at five or ten minute intervals, examine sections of the liver, to see what an enormous number of bacteria have been taken up by the endothelial cells and how these are in process of destruction. If we realise that this large number of cells of the body are capable of destroying bacteria, and that even virulent bacteria are liable to be taken up by those cells, and in many cases to be destroyed by them, we can understand there may be circulating, even in apparently healthy persons, virulent micro-organisms; and it only requires a special concatenation of circumstances—either great numbers of bacteria, or great virulence, or damage to epithelium, allowing a focus of disease to be set up in an individual spot—for it to set up trouble. We have to recognise, and we must realise, this destruction which is going on of virulent bacteria in the body throughout life; we can then begin to understand how it is that we get this curious irregularity in the deposition or localisation of tuberculosis in one or another case.

Professor MUIR, *Glasgow:* Mr. President and Gentlemen,—When I came here I had no intention of taking part in this discussion, but a number of questions have arisen of interest to pathologists upon which I should like to make one or two remarks. In the first place, I wish to express my high admiration for the splendid way in which the discussion was opened by the papers of Professor Benda and Professor Hamilton.

The subject before the meeting to-day has only two aspects, a theoretical and a practical. The theoretical concerns our work as pathologists and

Digitized by Google

teachers and expounders of the subject. There is one point which I think very striking with regard to the character of the discussion to-day, namely, that the description of distinct anatomical varieties of tubercle has largely been departed from. The characters of tuberculosis in the various organs have been looked at on broad general principles, and the most important of these is the mode of entrance and the distribution of the organisms. That is the first principle. Then, after that, in any particular place we have a lesion produced which depends, on the one hand, on the characters of the tissues, and on the other hand upon the virulence of the bacilli. I think there is no doubt whatever, as Dr. Habershon has said, that the virulence of the tubercle bacillus does vary, and very much so, in different cases. We see that in the characters of the different organs. Another example of this striking variation is seen in the number of bacilli which are present in the lesion. I used to think it possible to ascertain beforehand, from the characters of the lesions, what proportion of bacilli was likely to be found; but I now think it is quite impossible to do so.

With regard to the mode of spread, one point which I think was brought out by Professor Benda and Professor Hamilton was the spread of bacilli in a direction contrary to that of the flow of lymph. That, I think, is a very important point, and it is one which is recognised, but not so fully as it deserves to be. This is especially the case in pulmonary tuberculosis, because certain observers seem to assume that because tubercle is present in the lung therefore it is necessarily the result of inhalation. As a matter of fact, especially in young subjects, the bacilli very often spread from the glands of the lung into the lung substance; and the infection of the pulmonary tissue is not the primary lesion, but a secondary one. That, I think, is a matter of importance.

With regard to the distribution, I think the modes of distribution have been so fully dealt with by Professor Benda, as shown in this synopsis, that I need say very little. One mode which is apt to be overlooked is (*a*), under paragraph 11, the single metastasis which arises through occasional intrusion of bacilli into the blood stream.

With regard to the absence of local lesions, Professor Adami has made remarks in the direction which I would have taken up. In the case of these infections through the mucous surface, what appears to me to happen is something of this kind: Suppose bacilli are present in the milk and they are infective, then certainly they are absorbed through the mucous membrane. Now, in the mucous membrane, as has been pointed out, there may be no lesion, and very often there is no lesion. The occurrence of a lesion depends upon the condition of the lymph circulation to a large extent, and it also depends upon the manner in which the bacilli are seized upon by the cells. We will say no lesion occurs at the place of entrance; the bacilli are carried up along the lymph stream to the lymphatic glands, and they are almost immediately attacked by the phagocytes, as Professor Adami has said; and no doubt they are entirely destroyed in many cases by phagocytes. It is only when there is a summation of the supply—if I may so speak of it—of

Digitized by Google

bacilli time after time in the lymphatic glands that bacilli are able to gain the upper hand, and then we have a lesion, and subsequent caseation. On the other hand, when bacilli are present in very large numbers, for example, as in cases where there is phthisical sputum, you have a much more serious affection locally—a distinct ulceration.

There are various other theoretical points. With regard to tubercular pericarditis, I do not think it is a very common condition, but I have had an experience somewhat similar to that of Professor Adami. For some reason I have had a far greater number of cases in later years than I used to have. The disease not only occurs from time to time, but occurs in various forms, and that is a point which I think has not been sufficiently dwelt upon. Sometimes you have fibrinous exudations, which are difficult to recognise as tubercular, but with tubercles scattered throughout it. Sometimes there is great thickening of the layer of the pericardium, with caseous material between; and occasionally the amount of caseous change may be so very slight that the case appears to be one of obliterated pericardium. On careful looking there is only a small amount of caseous material to be seen on the base.

There is another form, namely, the hæmorrhagic tubercular pericarditis. In some cases the pericarditis has a distinctly hæmorrhagic character. What the reason of this is it is difficult to say, but in one case which I have had the opportunity of examining, the softness of the inflammatory material was marked and was studded over with large masses of tubercle bacilli.

The important thing for us, as pathologists, is to point out that the absence of a tubercular lesion in the intestine does not mean that bacilli have not entered through the mucous membrane, nor that the occurrence of a pulmonary lesion indicates that that lesion has been produced by inhalation. (Applause.)

Dr. BOUSFIELD: I rise with considerable diffidence, because I feel I have nothing whatever to add of my own to the information which has been given to us so abundantly this morning. Yet, on the other hand, representing, as I may perhaps say I do, the other class of workers in connection with tuberculosis—the people who are chiefly concerned in putting the theories and observations of pathologists into practical application for the protection of the community—I cannot help feeling that there are one or two points in connection with the discussion which has taken place which might usefully be brought together. It seems to me that the first point is—and that, I think, has been made abundantly clear, though not expressed in so many words—that there are two factors concerned in the conveyance of tubercular infection. The first of these is the virulence of the bacillus; and the second point is the susceptibility of the patient. With the susceptibility of the patient is bound up—and very importantly bound up, from my point of view—the question of the age of the patient as affecting that susceptibility. One hardly knows yet—and one is almost afraid to speak at the present moment—as to the influence of virulence of the bacillus in determining lesions which may result,

Digitized by Google

or determining the infection which may give rise to those lesions. This may be a little out of order, and yet the matter is emphatically germane to the subject with which we are dealing, to consider the bearing of Professor Koch's remarks the other day upon this subject. I think, as the subject of this discussion is not confined to human tuberculosis, I may be allowed to carry the matter a stage further, and to say that there appears to be very distinct evidence that we have, in regard to tuberculosis, at least three main classes of virulence. Regarding them all for the moment as identical, putting aside any consideration that there is a difference between human and bovine tuberculosis, we have the less virulent form which is represented by chronic pulmonary tuberculosis and other forms of corruption which we are accustomed to. As an illustration of the next stage of virulence there is the acute miliary tuberculosis produced by the ingestion of the organism. Finally, there is bovine tuberculosis, which leaves its victim completely riddled with tubercular deposits from one end of the body to the other. It is of the very last importance for us to have some means of guidance in dealing with these organisms as they may present themselves to us. And it is of very great importance to us that we should learn, in some way or another, to draw a distinction between the organisms which give rise to these classes of phenomena.

With reference to what was said by Professor McWeeney, I agree with him, personally, from my own observation. I think it is no heresy to say so, but there is a particular form of the tubercle bacillus which we have not yet learned to recognise; for instance, where the virulence of the organism is the most manifest, as in acute miliary tuberculosis of the lung, it is often exceedingly difficult to demonstrate the presence of the bacilli, and when found they are only in very small numbers indeed. And those who have examined the pus of a caseous suppurating gland know that it is with extreme rarity that the organism can be found, although the broken-down caseous material, when inoculated, is capable of producing a very distinct and decided affection of the animal inoculated.

I do not think I need detain the Section longer. Those were the main points that I wished to emphasise.

The CHAIRMAN: I think we have had the subject very fairly discussed this morning, and I have no doubt you will agree with me that we should like to hear what Professor Hamilton and Professor Benda have to say. Professor Benda will give us a demonstration upstairs of his sections and preparations, and we have yet three papers to get through. I will therefore call upon Professor Hamilton, and then Professor Benda; then, with your permission, we will adjourn for twenty minutes and then come back and hear papers.

Professor HAMILTON: Mr. President and Gentlemen,—I seem to have raised a hornet's nest about my head with regard to what I said about tubercular pericarditis. If there has been any subject raised this afternoon it

Digitized by Google

has been this one of tubercular pericarditis. But I venture to think that the honour which the various gentlemen have done me by contradicting what I stated regarding tubercular pericarditis is due in some part to a misunderstanding on their part. I perfectly agree with Professor Adami and others that a fair proportion of these cases, which to most of us appear to be inflamed pericarditis, when you come to examine them microscopically prove to be tubercular. But that is not the point I want to make out. I have exactly the same experience as Professor Adami; I ran out of material for my class, and I picked up what was supposed to be a simple case of pericarditis, and there, sure enough, were tubercles. That is not the point I want to make clear. It is this—and it is founded upon my work extending over thirty years—that in cases of acute eruption of tubercle all over the body, in children and others, where the pleura, the peritoneum, and the meninges may be the seat of acute tubercle, you may have the pericardium perfectly free, and there may be no pericarditis and no tubercle. I can substantiate that from my own cases, and cases of acute tuberculosis arising from a primary focus of infection where there is an acute eruption all over the body. I have kept notes of all these cases—I cannot tell you how many of them—for the last twenty or thirty years, and there are only four in which I have found acute irruption of tubercle upon the pericardium. I appeal to every pathologist in the room at the present time to say whether it is not his experience that where the lung is broken down—perhaps one gone from tuberculous ulceration and the other infiltrated with tubercle in process of softening—where the pleura is beset with tubercle, where every gland in the body and every tissue is riddled with tubercle, the pericardium may be entirely clear. That is the class of case I want to bring up. In the explanation of the complication of pericarditis with tubercle the question arises as to which is the cart and which is the horse. I have often thought that in these cases referred to in my paper the condition of immunity or partial immunity which the pericardium enjoys may be destroyed by incipient pericarditis, which may become tubercular afterwards. That is quite a possibility, and I think it may explain a certain number of these cases. I cannot believe that my experience is different from that of other pathologists; and what I wanted to make plain was that you may have every organ in the body tubercular but the pericardium may be clear, without a particle of tubercle upon it.

I am extremely obliged for the courteous manner in which you have received my remarks. They were hurriedly put together, but they have given rise to some discussion, and I think that discussion has been of a useful nature. We have listened to Professor Benda's paper with very great pleasure, and still more shall we be gratified by the demonstration which he will give us. I have already seen his beautiful specimens, which are worthy of everyone's careful notice.

Professor BENDA, in reply, said: Meine Herren,—Es ist in der Discussion eine solche Reihe von Fragen hier angeregt worden, dass die Zeit kaum ausreichen wird, um auf alle zu antworten. Es sind auch neue Punkte

Digitized by Google

herangezogen worden, die in dem einleitenden Referate nicht erwähnt worden sind, und wir können den Herrn Sprechern nur dankbar sein, dass sie den Gesichtskreis unserer Debatte in so hohem Masse dadurch erweitert haben. Indessen möchte ich auf einige dieser Einwände und Zusätze eingehen.

Ich gestehe vollständig den Herren Habershon, McWeeney, Sir C. Nixon und anderen zu, dass die Wichtigkeit der Virulenz des Tuberkelbacillus und der Aufnahmefähigkeit, der Widerstandsfähigkeit der Gewebe eine sehr grosse Rolle bei der Variation der Tuberculose spielt, und ich muss gestehen, dass ich auch sehr gerne auf diese Erörterung eingegangen wäre, wenn das Thema meines Referates nicht so formuliert gewesen wäre, dass die Pathologische Anatomie und Histologie zur Behandlung gestellt wurde. Die Virulenz der Bacillen und die Aufnahmefähigkeit der Gewebe können wir aber nicht mit einem Messer oder sonstigen anatomischen Hilfsmitteln erforschen.

Hinsichtlich der übrigen Fragen, freue ich mich, dass ich mit meinem hochverehrten Herrn Correferenten wohl einer Meinung in den meisten Punkten gewesen bin, und dass sich unsere Vorträge, obgleich wir leider nicht hatten in Beziehung treten können, ergänzten, so dass wir nicht beide genau dasselbe vor Ihnen zu sagen hatten.

Die meisten Differenzen habe ich mit Herrn Habershon. Es sind darunter einige Punkte, die ich besprechen muss. Zunächst war von mir nicht die Eingangspforte der Tuberculose erwähnt worden, aber ich bin sehr dankbar, dass hierauf die Discussion gelenkt worden ist. Ich bedaure, dass Herr Friedmann, der vorher anwesend war, das Lokal verlassen hat; derselbe war es, der vor einiger Zeit das Material der Berliner Krankenhäuser auf primäre Tonsillar-Tuberculose untersucht hat und mit vieler Mühe einige Fälle gefunden hat, bei denen er mit einiger Sicherheit primäre Tonsillar-Tuberculose festzustellen glaubte. In meinem Material habe ich keine primäre Tonsillar-Tuberculose gesehen. Ähnlich verhält es sich mit der primären Darm-Tuberculose. Dieser Punkt ist ja von Herrn Koch vorgestern besonders betont worden. Es sind dabei auch die Erfahrungen bedeutender Berliner Hospitäler mitverwandt worden, wie des Kaiser Friedrich Krankenhauses, das ein ungeheures Material von Kindersectionen hat, bei denen sich auch sehr wenig Fälle von primärer Darmtuberculose gefunden haben. Auch in meinem Material sind die Fälle ausserordentlich selten; ich bin aber überzeugt, dass ich solche Fälle gesehen habe, aber dass sie in unserem Material eine geringe Stelle einnehmen, kann ich versichern, ohne momentan statistische Zahlen dafür beibringen zu können.

Nun aber habe ich eine sehr erhebliche Differenz mit Herrn Habershon bezüglich der Verbreitung der Bacillen. Er will die Verbreitung durch die epithelialen Kanäle, die ich an erster Stelle genannt habe, in die letzte Stelle rücken und die Verbreitung durch die Lymphwege in die erste. Es giebt mir das Gelegenheit, was schon von McWeeney geschehen, auf die Milchdrüsen einzugehen, die ich vorher vergessen hatte. Auch dort sehen wir, dass die Epithelkanäle die Hauptverbreitungswege sind, wie das vorgestern durch Frau Dr. Rabinowitsch-Kempener angeführt worden ist. Die Fälle von Lungentuberculose aber, die Herr Habershon angeführt hat, wo nach Hämoptoë

Digitized by Google

plötzlich eine akute Verbreitung auf die Lungen beobachet worden ist, sind von uns auch gesehen worden, aber wir deuten sie anders: wir nehmen an, dass in diesem Falle durch das Blut Tuberkelbacillen aus den tuberculösen Geschwüren ausgespült und durch Inspiration in die Lungen weiter verbreitet worden sind. Ich glaube, dass diese Erklärung bedeutend wahrscheinlicher ist als dass durch die Hämoptoë eine Resorption in die Lymphwege stattgefunden hat.

Ob Tuberkelbacillen durch intacte Schleimhäute hindurchgehen, ist eine Angelegenheit, die pathologisch-anatomisch ausserordentlich schwer zu entscheiden ist. Wir werden dem Redner aber natürlich darin beistimmen, dass wir eine grosse Anzahl von Bronchialdrüsen-Tuberkeln finden, bei denen wir nicht eine Spur von Bronchial-Verletzungen finden; aber können wir daraus schliessen, dass die Tuberkelbacillen durch die intacte Schleimhaut hindurchgekommen sind? Wir haben neulich von Koch gehört, und wir wissen auch aus eigener Erfahrung, dass tuberculöse Läsionen vollständig heilen können, und wir können bei Kindern erwarten dass sie selbst ohne Narben heilen. Ist es da nicht möglich, dass die kleinen Läsionen, die die Tuberkelbacillen durchgelassen haben, vollständig geheilt sind? Ich halte diese Frage für eine gewisse Gefühlssache, für eine Liebhaberei eines jeden; ich glaube nicht, dass dadurch pathologisch-anatomische Beobachtungen zur Entscheidung kommen. Positive Beobachtungen sind wertvoller als negative darin und ich habe bisweilen nach langem Suchen vereinzelte Narben im Darm gefunden, wo der Darm anscheinend frei von Tuberculose war und nicht primäre Tuberculose des Darm sondern des Peritoneums und der Lymphdrüsen vorzuliegen schien. Es waren hier Narben nach Läsionen entstanden, die vielleicht im Stande gewesen sind, die Bacillen aufzunehmen.

Leider habe ich den Anfragen von McWeeney nicht ganz folgen können. Ich glaube verstanden zu haben, dass er zunächst eingewandt hat, dass er die metastatischen Eruptionen auf der Intima der Gefässe nicht beobachtet hat. Ich muss demgegenüber sagen, dass ich eine grosse Anzahl von mikroskopischen Präparaten beibringen kann, in denen sie zu sehen sind. Es gehört eine sehr grosse Uebung dazu, sie zu finden, und man muss erst einmal angefangen haben, sie zu finden, dann wird man erstaunt sein, wie oft man sie finden kann. Ich muss gestehen, dass ich vor 1894 noch keinen einzigen Gefässtuberkel gefunden habe, aber sobald ich einmal nach der Arbeit von Wild, in der Weigert angegriffen war, anfing diese Sache zu untersuchen, da war ich erstaunt, wieviel Tuberkel man fand und dass mar selbst in einem Falle, wo man zuerst nur einen fand, nachher unzählige andere fand, die man vorher nicht gesehen hatte. (Applause).

Ich kann nur, ohne den Beobachtungen irgend jemandes zu nahe treten zu wollen, die tröstliche Mittheilung machen dass auch in dem Berliner pathologischen Institut Virchow's sehr wenig Gefässtuberkel gefunden werden, und die Herren dort erstaunt waren, wenn ich alle paar Monate in der Berliner Medizinischen Gesellschaft wieder eine Gefässtuberculose zeigen konnte, obgleich wir das Material derselben Stadt bearbeiten.

Dann wurde die Frage gestellt, ob aus dem Blut unmittelbar Tuberkel-

Digitized by Google

bacillen durch die Drüsen hindurchgehen können. Diese Frage ist schon in früheren Jahren hinsichtlich der Erblichkeit der Tuberculose angeschnitten worden; sie hat auch in einer früheren Arbeit von Fani eine grosse Rolle gespielt für die Verbreitung der Bacillen durch das Sperma ebenso wie auch augenblicklich für die Infection der Milch. Mir scheint, dass niemals die intacten Drüsen den Tuberkelbacillus durchlassen. Die Untersuchungen von Herrn und Frau Kempener haben den Beweis erbracht, dass Tuberculose der Milchdrüse vorliegt, auch wenn wir sie makroskopisch nicht beobachtet haben. Ich habe durch die Güte der beiden Collegen ein einschlägiges Präparat aufstellen können, und daraus werden Sie sehen können, dass in einer Drüse, die auch ich makroskopisch für normal hielt, mikroskopisch nachweisbare schwere tuberculöse Läsionen sind in den Kanälen. An eine primäre Tuberculose der Epididymis glaube ich nicht, ich glaube aber, dass sie der Sitz einer solitären Metastase sein kann, wenn eine geringfügige primäre Tuberculose in einer Lymphdrüse vorhanden gewesen ist.

Sir C. Nixon hat die interessante Frage angeregt, wie es, ohne die Virulenz der Bacillen herbeizuziehen, erklärt werden kann, dass eine Heilung fieberhafter Tuberculosen vorkomme. Es ist ausserhalb meines Bereichs, über die Virulenz der Bacillen zu sprechen, aber ich kann wohl meinerseits das hervorheben, dass wir es auch pathologisch-anatomisch erklären können. Ich habe derartige Fälle in meinem Vortrage erwähnt, bei denen ich mikroskopisch nachweisen konnte, dass Gefässtuberkel geheilt sind. Diese Heilung kann auf verschiedene Weise erfolgen: erstens dadurch, dass die Gefässe thrombosieren, und dass der Tuberkel von der Blutcirculation abgeschlossen wird, dann aber auch dadurch, dass der Tuberkel vollständig organisiert wird, fibrös wird und dadurch heilt. Da ich die Symptome der sogenannten akuten Miliartuberculose ausschliesslich auf den Gefässtuberkel beziehe, glaube ich, dass wir in solchen Fällen wirklich von geheilten Attaquen der akuten Miliartuberculose werden sprechen können. Ich bitte Sie nochmals, in Hinblick auf diese Frage, die Präparate von dem thrombosierten Ductus thoracicus in Augenschein zu nehmen, die eine Erklärung dafür geben.

Damit glaube ich einen Teil der Fragen beantwortet zu haben. Meinem verehrten Freunde Professor Osler antworte ich nicht; ich bin beschämt, dass er mich derart gelobt hat; aber ich danke ihm für seine freundliche Gesinnung.

The PRESIDENT: We have three papers before us. Dr. Walsham cannot stay longer, and he therefore wished that his paper on "Excretion Tuberculosis of the Kidney" be taken as read. We have also a paper by Professor Ravenel, of Philadelphia. I think it would be well if Professor Benda were now to take the sections upstairs for twenty minutes, and then we could have these other two papers under discussion.

(Professor HAMILTON here presided.)

Professor RAVENEL (*Philadelphia*) read the following paper: "The

Digitized by Google

Comparative Virulence of the Tubercle Bacillus from Human and Bovine Sources":—

The relation existing between the various types of the tubercle bacillus found in man and in the lower animals has been the subject of much discussion for several years past, and studies of the morphology, biology, and virulence have been undertaken with the object of determining whether or not constant differences could be detected in the organism from these two sources sufficient to justify their classification as distinct species.

From a purely practical standpoint the question is narrowed to a study of the bacillus as found in man and in cattle, and in the determination of the relation they bear to each other. The importance of this study is immense, not only to the physician, anxious mainly for the protection of his clients, and to the general public, who look to the medical profession for guidance in such matters, but perhaps even more so to those who are concerned in the framing of laws and direction of public measures looking to the suppression of tuberculosis in man and animals. Efforts in this direction often meet with strong opposition, and extreme statements are made on very scanty evidence, which render the task of the hygienist much more difficult, and may result in the failure of his purpose. The lack of positive and authoritative knowledge in regard to this matter has in recent years led to the adoption of retrograde measures in several places. The problem is one that can be entirely cleared up only by a long series of observations and carefully conducted experiments, which are tedious, exacting, and require a considerable lapse of time before results can be had. The correct interpretation of these results is in many cases not an easy matter.

The identity of the tubercle bacillus, as found in the mammalia, went unquestioned for many years, and laws for the prevention of the transmission of the bacillus of cattle to man have been based mainly on this belief. In America, Dr. Theobald Smith was the first to take up the systematic study of the bacilli isolated from various of the lower animals and man, and to call attention to certain fairly constant differences observed in the organisms from the two sources, which he considers sufficient to justify classifying them as distinct varieties or races, though his experiments "show unmistakably the close relationship existing among the various cultures studied."*

Dr. R. R. Dinwiddie,† Pathologist and Bacteriologist of the Arkansas Agricultural Experiment Station (U.S.A.), has carried out quite extensive experiments, in which he has compared the virulence of the human and bovine tubercle bacillus, and also human tuberculous sputum with bovine material, for a number of the domestic animals, his results showing a greater power for both the bovine bacillus and material than for the human.

The vast practical importance of the matter, no less than its great scientific interest, was early realised by Dr. Leonard Pearson, State Veterinarian of Pennsylvania, and under his direction we have devoted much of our attention

* 'Transactions Association American Physicians,' 1896.

† 'Arkansas Agricultural Experiment Station,' Bulletin No. 57, June, 1899.

Digitized by Google

at the laboratory of the State Live Stock Sanitary Board, for more than two years past, to the study of the tubercle bacillus obtained from various cases of tuberculosis in man and in cattle.

In the carrying out of this work I have had throughout the efficient assistance of Dr. S. H. Gilliland, on whom a large share of the labour has fallen, and to whom is due much of the credit in its successful issue. To him, and to the several physicians who have interested themselves in obtaining material for us, I beg to express my obligation. During a considerable portion of the time that Part II. of the experiment was in progress, Dr. W. G. Shaw was in charge of the animals, and did much of the work; while the bovine material for the inoculations was obtained and prepared by Dr. J. J. Repp, both of whom were at the time connected with the laboratory.

Our experimental work has been divided into two parts :—

I. Isolation and study of pure cultures from various sources in man and cattle.

II. Testing the pathogenic power of tuberculous material of human and bovine origin.

I. Under the first division thirteen cultures, seven of human and six of bovine origin, are included in this report. Of these, two human and two bovine have been compared under conditions as nearly identical throughout as it was possible to obtain them. Cultures H (Bovine) and K (Human) were isolated within a few days of each other, while L (Bovine) and M (Human) were made on the same day. The first sub-cultures of both pairs were made on the same day, and all transfers since have been made simultaneously. The comparative pathogenicity of these cultures is shown in Tables I. and II.

Cultures H and K were tested also for puppies and pigs by feeding, as described below.

Culture H was recovered from the horse, goat, two puppies, two hogs, and man; while culture K was recovered only from two puppies and two hogs, the absence of lesions in other animals inoculated making it impossible to obtain a series comparable with culture H. The virulence of these recovered cultures, with the exception of that from the horse, was tested for guinea-pigs and rabbits, the results being given in Table III.

Human cultures I, J, U and W, and bovine cultures F, Q, QQ, and T, were isolated as occasion arose, and their virulence tested only for guinea-pigs and rabbits. The dose and mode of inoculation were uniform in all cases, and the animals were kept under the same conditions, but beyond this the tests are not entirely parallel. The results are given in Table IV.

In addition to those cultures here mentioned, we have isolated and examined nine others morphologically and culturally. These have been taken into account in forming our conclusions, as well as the very careful descriptions given by Dr. Theobald Smith of the cultures studied by him.

The terms "human" and "bovine" have been applied to cultures to denote their origin from man or from cattle. Each culture is designated by a letter of the alphabet, and the generation indicated by a small figure at the

Digitized by Google

lower right corner. Cultures which have been inoculated and recovered retain their original letter, a second letter enclosed in brackets being added to mark the recovered culture.

II. The second part of the work has been the inoculation of animals with tuberculous material from man and cattle. The plan and methods are described in another section, while the results are given in Tables V. and VI. As a part of this experiment, human tuberculous sputum has been fed to two calves, and four have been inoculated with the same material. These have been included in Table V., and described further on.

Part I.—Isolation and Study of Pure Cultures from Various Sources in Man and Cattle.

Method of isolating pure cultures.—The isolation of cultures has been obtained by the method devised by Dr. Theobald Smith,* the principal features of which are: (1) The test-tubes, which are fitted with a cap like that of the well-known Miquel flask, ground on. The small tubulation in the cap is plugged with glass wool. (2) The medium, which is dog's serum. The animal is bled with the strictest aseptic precautions, and the blood conducted to sterile jars through sterile rubber tubes. The serum is drawn off with pipettes and distributed at once into the test-tubes, put in the hardening oven, and coagulated at a low temperature (76° C.). Prepared in this way the serum needs no sterilisation, and is much softer than when a higher temperature is employed. (3) The tubes are kept always inclined. (4) The tuberculous tissue selected for planting in our culture tubes is not crushed, nor rubbed over the surface, as advised in other methods. Young nodules from the omentum, spleen, liver, or a lymphatic gland are cut out, and a block of considerable size is placed on the surface of the serum. After remaining from two to three weeks in the incubator at 37·5° to 39° C., the tissue is pressed against the sides of the tubes with a stout platinum wire, and rubbed over the surface of the medium. Examination of the fluid squeezed out of the tissue at this time will generally indicate the final result. The tubes are replaced in the incubator, and within a week or ten days, colonies of the tubercle bacillus usually appear. The nodules for inoculation of the tubes are best obtained from guinea-pigs, which are to be inoculated with the original matter from which we wish to obtain cultures. As soon as these show marked illness they are chloroformed, and the cultures made. In general terms, the more recent the lesions the greater the chances of successful culture. As a rule, guinea-pigs can be killed in from three to four weeks after inoculation. If our material is of human origin the inoculation should be intra-peritoneal; if bovine, subcutaneous inoculation will generally cause a sufficiently rapid involvement of the organs. (5) The incubator should contain a dish of water, to insure an abundance of moisture in the atmosphere. With the same object in view, it should be opened as seldom as possible.

* 'Journal of Experimental Medicine,' Vol. III., Nos. 4 and 5, 1898.

Digitized by Google

The only modification of the above method in our work has been the addition of a 50 per cent. solution of glycerine in water to the serum, in such proportion that the resulting mixture contains 5 per cent. of glycerine. This seems to make coagulation slightly more tardy, but the serum does not dry out as quickly, and the growth of the tubercle bacillus is facilitated. Serum prepared in this manner has been employed throughout for the cultures used in our inoculations.

Preparation of cultures for inoculation, and dosage.—The course of an experimental tuberculosis depends so largely on the number of bacilli introduced that exact dosage is an important factor in comparative work. Weighing naturally suggested itself, but a trial soon convinced us of its impracticability. The growth of human cultures is usually so abundant by the second generation, that it is easy to obtain fairly dry masses which can be weighed quite accurately, but bovine cultures often grow very scantily for many generations, as a thin film resembling ground glass. We have adopted as a uniform practice the method of Dr. Th. Smith—the use of a suspension of a given opacity. A portion of the growth is removed to the wall of a dry and sterile test-tube, and rubbed round and round with a glass rod until no lumps can be seen. Bouillon is then added, and, after stirring thoroughly, the mixture is allowed to stand quiet for two or three hours, so that the clumps may settle. The upper portion is poured off, and bouillon added until the suspension equals in turbidity a twenty-four-hour-old culture of the typhoid bacillus. The test culture is killed with formalin vapour and sealed, so that it can be used as a standard for a considerable time. Cover-glass preparations were made always as controls. In the tables below the generation of the cultures is indicated by the exponent, and the age of each culture is given in days in a separate column. For guinea-pigs and rabbits, the dose has been throughout 1 cc. of the suspension, introduced under the skin of the abdomen. For the other animals the dose and mode of inoculation are given in the tables.

Morphology and Cultural Characteristics of Bacilli Examined.

The morphology of the bacilli in cultures of bovine origin is more uniform and constant than in cultures from man. The bovine bacilli are short, seldom more than 2 microns in length, and averaging less. In early generations many are seen which are oval, their length not more than double their breadth. They are thick and straight. They stain with carbol-fuchsin evenly and deeply, and beading is markedly absent, even in old cultures.

The human bacilli are, as a rule, much longer from the start, and tend to increase in length rapidly in sub-cultures. They are generally more or less curved, and some cultures contain many S-shaped forms, as culture M noted below. They stain with carbol-fuchsin less deeply, and beading is a marked characteristic, often seen in the earliest growth.

These characteristics are most persistent in cultures on blood serum. On

Digitized by Google

glycerine agar, glycerine bouillon and potato with glycerine, the bacilli from the two sources approach each other in appearance and morphology much more closely.

The human cultures isolated have without exception grown more luxuriantly than the bovine cultures, though in at least two instances (cultures U and BB) the first growth was very long in appearing. This was due, no doubt, to their feeble virulence and consequent slow production of lesions in the tissues of guinea-pigs. A saprophytic life once established, they grow vigorously.

The bovine cultures are apt to grow as discrete colonies in the first culture, and for several generations are likely to grow as an exceedingly thin layer over a part of the medium, resembling closely ground glass.

The human bacillus can usually be induced to grow on glycerine agar in sub-cultures made from the original growth on blood serum. All attempts to obtain a like result with the bovine organism have failed.

Three of the bovine cultures examined have been isolated from milk. Two of them, L and Q, have shown throughout the characters of the type described as "bovine," while T approaches the human bacillus in morphology, being long and slender. It is, however, more virulent than any human culture tested.

Among the human cultures only one (M) has been isolated from sputum. Through the kindness of Dr. Alfred Hand, Pathologist of the Children's Hospital, to whom we beg to make grateful acknowledgments, we have been able to obtain cultures from three cases of tuberculosis in young children, in which the intestine was involved. One of these, from which culture BB was isolated, was considered by Dr. Hand to be "more clearly than any other he had ever seen, of intestinal origin," and was, therefore, studied with peculiar interest. Its pathogenicity has not yet been determined, but the course of the disease in the guinea-pigs inoculated to obtain the culture indicated a very feeble virulence, one living sixty-six and another ninety-six days. The bacilli, even in the first generation, are unusually long and thick. They stain deeply with carbol-fuchsin, but are beaded in a striking manner; the brightly stained portions being quite regularly disposed along the rod. They are most unlike what we have described as the bovine type in every way.

Cultures U and W, also from children with intestinal lesions, correspond to the human type in every particular, both as regards morphology and virulence.

Comparison of Cultures H (Bovine) and K (Human).

History of Cultures.

Culture H (Bovine) was isolated from the mesenteric gland of a Jersey Grade cow about seven years old, which had been slaughtered for beef, but was found to be extensively diseased, and condemned. The lungs were the seat of an extensive and long-continued tuberculosis. Most of the foci had undergone caseation. The liver was involved also, but to a less extent. The gland from which the culture was obtained had undergone cheesy degenera-

Digitized by Google

TABLE No. I.

TABLE SHOWING VIRULENCE OF BOVINE CULTURE H.

Culture.	Age in days.	Animals.	Date of Inoc.	Method of Inoc.	Dose in c. c.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Died.	Killed.	Length of Life.	Average.
H_2	15	Guinea-Pig	Sept. 2	Subcutaneous	1	290 grms.	174 grms.	116 grms. loss	Sept. 25		23 days	
,,	15	,,	,, 2	,,	1	310 ,,	224 ,,	86 ,, ,,	Oct. 9		37 ,,	$28\frac{1}{3}$ days
,,	15	,,	,, 2	,,	1	300 ,,	200 ,,	100 ,, ,,	Sept. 27		25 ,,	
,,	15	Rabbit	,, 2	,,	1	1730 ,,			Nov. 7		66 ,,	
,,	15	,,	,, 2	,,	1	1580 ,,			Oct. 19		47 ,,	$56\frac{1}{2}$,,
,,	29	Dog	,, 16	Intra Pulmon.	2	20 lbs.	18 lbs.	2 lbs. ,,	Nov. 14		59 ,,	
,,	29	,,	,, 16	,,	2	60 ,,			,, 21		66 ,,	$62\frac{1}{2}$,,
H_3	73	Horse	Dec. 9	,,	14	1030 ,,	990 ,,	40 ,, ,,		June 25, 1900	6 m. 16 d.	
,,	73	Goat	,, 9	,,	4	79 ,,	61 ,,	18 ,, ,,	Dec. 31		22 days	
,,	73	,,	,, 9	,,	4	29 ,,	$20\frac{1}{4}$,,	$8\frac{3}{4}$,, ,,	Jan. 4, 1900		26 ,,	24 ,,

TABLE SHOWING VIRULENCE OF HUMAN CULTURE K.

Culture.	Age in days.	Animals.	Date of Inoc.	Method of Inoc.	Dose in c. c.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Died.	Killed.	Length of Life.	Average.
K_2	15	Guinea-Pig	Sept. 2	Subcutaneous	1	380 grms.	270 grms.	110 grms. loss	Oct. 14		42 days	
,,	15	,,	,, 2	,,	1	320 ,,	175 ,,	145 ,, ,,	Sept. 30		28 ,,	38 days
,,	15	,,	,, 2	,,	1	310 ,,	215 ,,	95 ,, ,,	Oct. 16		44 ,,	
,,	15	Rabbit	,, 2	,,	1	1375 ,,	1970 ,,	595 ,, gain		May 25, 1900	8 m. 23 d.	
,,	15	,,	,, 2	,,	1	1500 ,,	1820 ,,	320 ,, ,,		,, 25, 1900	8 m. 23 d.	8 m. 23 d.
,,	29	Dog	,, 16	Intra Pulmon.	2	21 lbs.	20 lbs.	1 lbs. loss		Apr. 20, 1900	7 m. 4 d.	
,,	29	,,	,, 16	,,	2	57 ,,	54 ,,	3 ,, ,,		Mar. 19, 1900	6 m. 3 d.	6 m. 19 d.
K_3	79	Horse	Dec. 9	,,	14	1275 ,,	1370 ,,	95 ,, gain		June 25, 1900	6 m. 17 d.	
,,	79	Goat	,, 9	,,	4	73 ,,	75 ,,	8 ,, ,,		,, 29, 1900	6 m. 21 d.	
,,	79	,,	,, 9	,,	4	28 ,,	37 ,,	9 ,, ,,		,, 29, 1900	6 m. 21 d.	6 m. 21 d.

Digitized by Google

tion. On May 17, 1899, guinea-pigs were inoculated intra-peritoneally with an emulsion made from the centre of this gland. On July 6, one pig was killed, and cultures made on dog's serum. On July 28, the first growth was observed, and on August 18 sub-cultures were made from the omentum. Growth was fairly abundant. The bacilli were short, thick, straight, and stained evenly and deeply.

Culture K (Human) was isolated from the lung of an adult negro man. The disease was not severe and was confined almost entirely to the lungs. Death was due to heart trouble. Guinea-pigs were inoculated intra-peritoneally with an emulsion made from nodules taken from the lungs on June 10, 1899. On June 28, a pig was killed and cultures made on dog's serum. Growth was first observed on July 29, the cultures from the spleen being used for sub-cultures, the first of which was made on August 18. This culture grew rapidly and abundantly from the beginning. The bacilli were quite long, slender, and curved. They stained somewhat faintly, and showed marked tendency to beading.

Comparison of Cultures L (Bovine) and M (Human).

History of Cultures.

Culture L (Bovine) was obtained from the milk of a cow with tuberculosis of the udder. On May 22, 1899, a guinea-pig was inoculated with 10 c.c. of this milk. It died July 8, and from it a second guinea-pig was inoculated intra-peritoneally with an emulsion made from the spleen and omentum. On August 25 this pig was killed, and two others inoculated intra-peritoneally with an emulsion made from the spleen and omentum. One of these was killed on September 18, and cultures made on dog's serum. On October 20 growth was first observed. On November 17 sub-cultures were made. The growth for several generations was very scanty. The bacilli were extremely short, many oval in shape. They took the stain evenly and deeply.

Culture M (Human) was obtained from the sputum of a young adult female. Throat symptoms were most marked in this case. The disease was rapid and violent, death occurring in about five weeks after the sample of sputum was obtained. It contained large numbers of tubercle bacilli. On July 12, 1899, a guinea-pig was inoculated subcutaneously with the sputum. It was killed August 25, and two pigs inoculated intra-peritoneally with an emulsion made from the spleen and omentum. On September 18, a pig was killed and cultures made on dog's serum. Growth was first observed on October 15, and sub-cultures were made on October 17. Growth was rapid and abundant from the first, and the first transfers made from the original culture to glycerine agar grew luxuriantly. The bacilli in the original culture were rather short, irregular in shape, many S-shaped and as a rule stained quite evenly, and deeply. In sub-cultures, the bacilli soon became much longer, straighter, and showed beading.

Digitized by Google

TABLE No. II.

Table Showing Virulence of Bovine Culture L.

Culture.	Age in days	Animals.	Date of Inoc.	Method of Inoc.	Dose in c. c.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Died.	Killed.	Length of Life.	Average.
L_2	35	Guinea-Pig	Dec. 22	Subcutaneous	1	330 grms.	210 grms.	120 grms. loss	Jan. 13, 1900		22 days	26⅜ days
,,	35	,,	,, 22	,,	1	424 ,,	240 ,,	184 ,, ,,	,, 13, 1900		22 ,,	
,,	35	,,	,, 22	,,	1	286 ,,	246 ,,	40 ,, ,,	,, 27, 1900		36 ,,	
,,	35	Rabbit	,, 22	,,	1	1720 ,,	1216 ,,	504 ,, ,,	Mar. 27, 1900		95 ,,	96½ ,,
,,	35	,,	,, 22	,,	1	2100 ,,	1450 ,,	650 ,, ,,	,, 30, 1900		98 ,,	
,,	35	Dog	,, 22	Intra Pulmon.	3	31 lbs.	30½ lbs.	1½ lbs. ,,		Apr. 30, 1900	129 ,,	130 ,,
,,	35	,,	,, 22	,,	3	25½ ,,	27 ,,	1½ ,, gain		May 2, 1900	131 ,,	
,,	36	Goat	,, 23	,,	4½	83 ,,	73 ,,	10 ,, loss	Jan. 8, 1900		16 ,,	

Table Showing Virulence of Human Culture M.

Culture.	Age in days.	Animals.	Date of Inoc.	Method of Inoc.	Dose in c. c.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Died.	Killed.	Length of Life.	Average.
M_2	35	Guinea-Pig	Dec. 22	Subcutaneous	1	350 grms.	260 grms.	90 grms. loss	Feb. 14, 1900		54 days	34 days
,,	35	,,	,, 22	,,	1	410 ,,	300 ,,	110 ,, ,,	Jan. 13, 1900		22 ,,	
,,	35	,,	,, 22	,,	1	300 ,,	216 ,,	84 ,, ,,	,, 17, 1900		26 ,,	
,,	35	Rabbit	,, 22	,,	1	1620 ,,	1740 ,,	120 ,, gain		May 25, 1900	154 ,,	154 ,,
,,	35	,,	,, 22	,,	1	1730 ,,	1930 ,,	200 ,, ,,		,, 25, 1900	154 ,,	
,,	35	Dog	,, 22	Intra Pulmon.	3	29½ lbs.	22 lbs.	7½ lbs. loss		Apr. 27, 1900	126 ,,	92½ ,,
,,	35	,,	,, 22	,,	3	21 ,,	18½ ,,	2½ ,, ,,	Feb. 19, 1900		59 ,,	
,,	36	Goat	,, 23	,,	4½	52 ,,	48 ,,	4 ,, ,,		June 29, 1900	6 m. 6 d.	

Digitized by Google

Summary and Post-Mortem Notes.

It will be seen from the above tables that for each culture ten animals were used. Of the ten inoculated with culture H (bovine), nine died, while only one had to be killed, this being the horse; while of those inoculated with culture K (human), only three died, while seven had to be killed. With each of cultures L (bovine) and M (human), eight animals were inoculated. Of those inoculated with culture L, six died and two were killed; while for culture M four died and four were killed. Taking the totals of these four cultures, eighteen animals were used for the bovine cultures and eighteen for the human. Of those inoculated with bovine tubercle bacillus, fifteen died and three were killed; while of those inoculated with the human cultures, seven died and eleven were killed. By making a closer study of the tables further differences in virulence between the bovine and human cultures may be brought out. All the guinea-pigs inoculated with these four cultures died, the extent of the post-mortem lesions being practically identical in all of them. Those inoculated with the human culture K lived almost ten days longer than those inoculated with the bovine culture H; while those inoculated with human culture M lived 7 1–3 days longer than those inoculated with bovine culture L. All the rabbits inoculated with both of the human cultures had to be killed and showed no post-mortem lesions whatever, while those inoculated with the bovine cultures died, with extensive lesions. These lesions varied somewhat in the different animals, necrotic areas at the point of inoculation, and complete involvement of the lungs being common to them all. The kidneys were also generally involved. Only one animal had generalised tuberculosis, involving the lungs, liver, kidneys, spleen and omentum.

Dogs.—The dogs showed a varying degree of susceptibility to all the cultures. Both of those inoculated with bovine culture H died, one in fifty-nine days, the other in sixty-six days. The smaller of the two had shown signs of illness for some days before death. The point of inoculation was well marked in the lung by a mass of tubercles 2·5 cm. in diameter. Both lungs were infiltrated throughout with gray nodules from 2 mm. to 4 mm. in diameter. Both pleural cavities contained a considerable amount of purulent effusion. The right pleura was roughened, without adhesions. The outer surface of the pericardium was thickly sown with minute nodules about 2 mm. in diameter, its interior being smooth. The liver was infiltrated throughout, and necrotic, giving a nutmeg appearance. The right kidney contained a number of tubercular nodules; the left was congested, but otherwise normal. The spleen had escaped infection. Through an accident no post-mortem was obtained of the other dog, but from his symptoms for some days before death it is reasonably certain that the disease was general, affecting particularly, perhaps, the lungs. Both of the dogs inoculated with human culture K had to be killed, the lesions in both being insignificant. In one the only marked change which could be found corresponded to the point of inoculation in the lung, where a small excavation was found almost completely filled in with scar tissue,

Digitized by Google

radiating from the centre in star shape. Scrapings from cut surfaces of this area revealed the presence of tubercle bacilli. All other organs were entirely normal. In the other dog the point of inoculation was marked by a large nodule in the pleural surface of the lung. The surfaces of both lungs showed a great number of minute nodules. Scrapings from cut surfaces revealed tubercle bacilli in small numbers. On the external surface of the pericardium was a nodule about one inch in diameter, which contained calcareous foci, incapsulated in dense fibrous material. The mediastinal glands were enlarged and contained a number of caseous foci. In all other respects the animal seemed normal.

Horses.—A marked difference was noted in the effect of the cultures on the horses. The horse inoculated with bovine culture H showed no ill effects from the injection for some months. In the latter part of March, nearly four months after the injection, some emaciation could be noted, and the respiration was quickened, being on an average of 22 a minute and somewhat laboured. On the 1st of April the temperature averaged 102·2° F. The symptoms grew very slightly more marked, and on June 25, six months and a half after inoculation, the animal was killed. It had lost 40 pounds in weight, though its condition was still fair. The costal pleuræ were extensively covered with masses of nodules of from 2 mm. to 18 mm. in diameter, averaging nearly 12 mm. in thickness. These growths were somewhat more extensive on the left side than on the right, and tended to be more isolated and somewhat larger in size. The lungs were a grayish pink, the blood-vessels being plainly outlined in a delicate network over the surface. They were covered with nodules, varying from 1 mm. up to 6 mm. in size, and closely packed. Both lungs were hard, dense, and devoid of elasticity, having much the feel of liver. At the point of inoculation in the right lung a small depression was found, filled in with a star-shaped cicatrix. This cicatricial tissue was surrounded with a nodule about 2·5 cm. in diameter, containing cheesy areas and much fibrous tissue. In the posterior part of the left lung a tubercular abscess about 2·5 cm. in diameter was found, and in the bottom of the lung several similar in size and character were seen. The bronchial glands were enlarged to twice their normal size, but showed no caseation. The surface of the pericardium was studded with minute nodules. The thoracic surface of the diaphragm was covered over a large portion of its surface with nodules the size of a pea, some of them pedunculated. Among the nodules were many shreds of fibrine, some of them 2·5 cm. in length. Similar masses were seen also on the costal surface of the lung. The liver, spleen, and kidneys appeared normal.

The horse inoculated with human culture K showed no clinical evidence of injury. The 1st of April its respiration was 12 a minute, its temperature was 100·5° F. It was killed on June 25, about six months and a half after inoculation. Its condition was good. It had gained 95 pounds in weight. There was considerable fat subcutaneously, and about the abdominal organs. All of the abdominal organs were entirely normal, with the exception of the liver, on the surface of which many fringes of fibrine were found. In the thoracic cavity, both pleuræ were smooth and shiny, except at the point of

Digitized by Google

inoculation on the right side, where a mass of small nodules, with fringes of fibrine, were seen. At the point of inoculation in the right lung was a cheesy abscess 4 cm. in diameter, and incapsulated with dense fibrous tissue, the walls being 6 mm. thick. Otherwise, the lung was entirely normal. In the left lung were three small tuberculous nodules about 12 mm. in diameter, situated near the surface, and which had undergone caseation. The disease was evidently non-progressive, and was probably retrogressive.

Goats.—Both the goats inoculated with bovine culture H died, one in twenty-two days and the other in twenty-six days, the lesions being much the same in both. There was thickening of the pleuræ and adhesion at the point of inoculation; otherwise they were normal. The lungs were covered with myriads of minute tubercles about 1 mm. in diameter, which gave a gritty feel to them. In the substance of the lung no tubercles could be found. The cut surfaces presented a pneumonic appearance, most marked in the smaller of the two animals. Scrapings from cut surface in any portion of the lung revealed enormous numbers of tubercle bacilli. The mucus in the bronchial tubes throughout the lungs also contained large numbers of tubercle bacilli. In the larger animal the liver, spleen, kidneys, peritoneum, and diaphragm were normal. The animal lost 18 pounds in weight in the twenty-two days of its life after inoculation. The smaller goat showed involvement of the abdominal organs also. The omentum was studded with pearly tubercles 2 mm. in diameter, one of which had undergone caseation. The surface of the spleen was thickly covered with tubercles 1 mm. in diameter. In the substance of the spleen no nodules could be found, but scrapings from cut surfaces showed a large number of tubercle bacilli. The liver showed no macroscopic tubercles, but scrapings from cut surfaces were rich in bacilli, and in sections under the microscope tubercles were seen, with a few giant cells. Microscopic examination of the lungs, however, revealed no tubercles. The infection in this animal was diffuse, and it is considered as a case of tubercular septicemia.

Both of the goats inoculated with human culture K gained in weight, and were killed after six months and twenty-one days. Both were in good condition, having a considerable amount of fat in the subcutaneous tissues and around the abdominal organs. In the larger animal at the point of inoculation there was an area of adhesion to the pleura about 8 mm. in diameter; on the surface of the left lung were two or three nodules about 3 mm. in diameter which had undergone caseation. This lung is attached at its lower edge to the diaphragm by fringes of fibrine, and along this edge a few nodules about 12 mm. in diameter, and containing cheesy pus, were found. The entire surface of the left lung was covered with fringes of fibrine of a reddish pink colour. In both lungs the elasticity was preserved and neither revealed nodules either by palpation or on section. The left pleura was covered with a network of fibrine similar to that seen in the lung; the right was normal. The mediastinal glands were much enlarged and calcareous. In the liver two small nodules were found near the surface, the organ being otherwise normal. In the smaller animal the omentum contained upwards of a hundred nodules from 2 to 12 mm. in diameter. The left lung was attached to the pleura at

Digitized by Google

its edge by numerous bands of fibrine, and at its posterior surface to the diaphragm in the same manner. Along the lower border were a number of nodules about 6 mm. in diameter, which had undergone caseation. The right lung was normal. The mediastinal glands were enlarged and calcareous. The left pleura showed ten or twelve nodules 6 mm. in diameter, which were cheesy; besides a large one, corresponding to the point of inoculation. The diaphragm was attached to the spleen and to the stomach by bands of fibrine, but no nodules were found. The liver was normal; the portal glands were enlarged and contained many cheesy nodules. The left kidney contained a number of cheesy nodules. The right kidney normal. The mesenteric glands were enlarged and several of them contained calcareous areas.

Comparative Virulence of Bovine Culture H and Human Culture K for Puppies and Pigs when Introduced Through the Alimentary Tract.

Puppies.—Four puppies eight weeks old, from the same litter, were selected, two of which received a suspension in milk of bovine culture H, and two a similar suspension of human culture K, equal amounts being given on ten days, six days in succession, with an interval of seven days, then four days in succession. The regular food was sterilised milk, bread and boiled beef.

Only one of the four puppies died, one which received the human culture K, death occurring after fifty-seven days. There was a generalised tuberculosis most marked in the lungs and liver. The other puppy was killed after eighty-five days. The only lesions found were small cheesy nodules on surface of one lung, in parotid and mesenteric glands. The two puppies fed with bovine culture H showed no ill effects whatever, and were killed on the eighty-fifth and eighty-seventh days respectively. Both were in good condition, the only lesions found being in the parotid and mesenteric glands, which contained minute caseous areas, none larger than 2 mm. in diameter.

Pigs.—As with the puppies, four pigs from the same litter, eight weeks old, were taken, two being fed with human culture K, and two with bovine culture H, equal amounts of a suspension in sterile water being soaked into bread, and given for ten days, with an intermission of only one day. The regular food was sterilised milk, ground oats, bran and corn meal.

All four of the pigs died, the average life of the two fed with human bacilli being 118½ days as against 100½ days for the two which received the bovine culture. One pig of each pair lived 123 days, dying on the same day. The total difference in gain of weight was only half a pound. All showed post mortem a generalised tuberculosis. While some differences were noticed in the extent of the disease in various organs, it cannot be said that these were such as to indicate a greater virulence of one culture than the other. In all the mesenteric glands were extensively diseased, while in one only, fed with bovine bacilli, was there ulceration of the mucous membrane of the intestine. Three of the four showed tuberculous ulceration of the tonsils. The remaining

Digitized by Google

TABLE SHOWING VIRULENCE OF E

Culture.	Recovered from.	Age in days.	Animal
H $(A)_3$	Goat	22	Guinea-
,,		22	,,
,,		22	,,
H $(B)_2$	Puppy	42	,,
,,		42	,,
,,		42	,,
H $(C)_2$	,,	41	,,
,,		41	,,
,,		41	,,
H $(D)_2$	Hog	36	,,
,,		36	,,
,,		36	,,
H $(E)_2$	Man (S. H. G.)	27	,,
,,		27	,,
,,		27	,,
H $(F)_2$	Hog	38	,,
,,		38	,,
,,		38	,,
H $(A)_3$	Goat	22	Rabbi
,,		22	,,
H $(B)_2$	Puppy	42	,,
,,		42	,,
H $(C)_2$	,,	41	,,
,,		41	,,
H $(D)_2$	Hog	36	,,
,,		36	,,
H $(E)_2$	Man (S. H. G.)	27	,,
,,		27	,,
H $(F)_2$	Hog	38	,,
,,		38	,,
K $(B)_2$	Puppy	39	Guinea-
,,		39	,,
,,		39	,,
K $(C)_2$	,,	42	,,
,,		42	,,
,,		42	,,
K $(D)_2$	Hog	29	,,
,,		29	,,
K $(E)_2$	,,	38	,,
,,		38	,,
,,		38	,,
K $(B)_2$	Puppy	39	Rabbi
,,		39	,,
K $(C)_2$	,,	42	,,
,,		42	,,
K $(D)_2$	Hog	21	,,
,,		21	,,
K $(E)_2$	,,	38	,,
,,		38	,,

Digitized by Google

Digitized by Google

one presented no gross changes in this gland, but microscopically areas of necrosis, apparently in the lymph follicles, were found, and sections stained with carbol-fuchsin revealed large numbers of tubercle bacilli. The two pigs which lived longest, one bovine and one human, both had extensive involvement of the joints, so that for several weeks before death walking was almost impossible.

The result of this experiment is inconclusive as far as the puppies are concerned, except as showing that they are readily infected through the alimentary tract. The pigs showed practically an equal susceptibility to the two cultures. The ease with which they were infected, and the extent of the lesions produced in so short a time, were both most striking features, and should serve to put farmers on guard against feeding their swine on milk from tuberculous cattle. In Pennsylvania we have observed several instances in which neglect of this precaution has led to considerable loss.

The cultures included in Table III. were all recovered from animals which had been inoculated or fed with cultures H and K. Their virulence was tested only for rabbits and guinea-pigs. The most striking thing to be noticed in the results is the increase in virulence in the human bacillus recovered from the hogs, K (D) and K (E). The average length of life for guinea-pigs after inoculation with the original culture K was thirty-eight days, while the rabbits were killed after eight months and twenty-three days, showing successfully resisted infection. On the other hand, the culture recovered from the hogs killed guinea-pigs in twenty-nine and a half days and thirty-two and a third days, and rabbits in seventy-one and a half and sixty-two and a half days respectively, showing a virulence for these animals which corresponds to that seen in bovine cultures.

Culture H (E) was recovered from a nodule on the hand of Mr. S. H. G., who accidentally inoculated himself while making a post-mortem on one of the goats which had succumbed to the original culture H. Particular interest attaches, therefore, to this culture. No marked difference can be detected in the virulence of this culture as compared with the original.

Considerable difference has been noted in the readiness with which the recovered cultures grew in the early generations. H (A), H (B), H (D), and H (F) all grew vigorously from the start, the three last growing without any stirring of the tissue on the original tubes, while H (G) grew on tubes sealed with paraffin, a result which I have been able to obtain only with the human bacillus, with this exception. The morphology of all the recovered cultures corresponded closely to the original.

Comparison of other Human and Bovine Cultures.

History of Cultures.

Culture F (bovine).—Obtained from a large tumour on the diaphragm of a cow which had been slaughtered for beef. The tumour was 13 cm. long, and from 7 to 9 cm. in diameter. On the surface were a number of grape-like masses of pearly lustre. On section numerous caseous areas were found,

Digitized by Google

from which an emulsion was made. It contained many tubercle bacilli. A guinea-pig was inoculated intra-peritoneally May 16, 1899. Death occurred on June 8, and from tissues a second pig was inoculated intra-peritoneally. This pig was chloroformed July 31, and a third pig inoculated in the same manner, which was chloroformed August 21, and cultures made from spleen and omentum. Scanty growth was observed on September 27. Under the microscope the bacilli were thick, short, and showed little tendency to beading, staining deeply and uniformly. The further history of this culture is given in the paragraph on "Retention of Characteristics in Culture."

Culture Q (bovine).—Obtained from the lungs of cow No. 5986. This cow was brought to our department on July 6, 1898, for experimental purposes, being considered then an advanced case of tuberculosis. She was emaciated and coughing badly. Many tubercle bacilli were found in the mucus she coughed up, which were long, thin, and much beaded. She died February 12, 1900. The lungs were infiltrated throughout, many of the areas having undergone caseation and calcification. It was difficult to understand how the animal lived as long as it did. The mediastinal glands formed a tumour 35 cm. long by 15 cm. in diameter, which was cheesy on section. The mesenteric glands were enlarged and cheesy. A guinea-pig was inoculated in the peritoneal cavity on November 15, 1899. On November 30 the animal was killed and cultures made. Growth was observed first on January 10, and sub-cultures made on the same date. Growth was always scanty.

Culture QQ (bovine).—Obtained from the mesenteric gland of cow No. 5986. An emulsion from the centre of the gland was inoculated into the peritoneal cavity of a guinea-pig February 13, 1900. On March 5 it was chloroformed and cultures made. Growth was observed on April 12, and sub-cultures made on April 17. Growth was more abundant than that of the preceding cultures (Q), but stlll scanty.

Culture T (bovine).—Obtained from milk sent in for examination by a dairyman in Philadelphia. It was inoculated into the peritoneal cavity of a guinea-pig January 24, 1900. The guinea-pig was chloroformed on April 6. With an emulsion of its spleen two other guinea-pigs were inoculated intra-peritoneally on the same date. One of these was chloroformed on April 28 and cultures made. Growth was observed first on June 23, and cultures made the same day. Growth was abundant and luxuriant.

Culture U (human).—Obtained from a child three years of age, whose death was due to tubercular meningitis. Autopsy. Lungs and bronchial lymphatic glands full of miliary tubercles. Anterior mediastinal glands much enlarged. Spleen extensively involved, liver less so. On abdominal surface of diaphragm were a number of flat, yellow nodules. Mesenteric glands enlarged, and contain old, yellow, cheesy nodules. One small tubercle found in right suprarenal. Purulent and cheesy nodules found in meninges and encephalon. The culture was obtained from mesenteric glands.

The pathologist, Dr. Hand, was uncertain as to the origin of this case.

Guinea-pigs were inoculated intra-peritoneally on June 15, 1900. One was killed on July 23, and with its tissues a second was inoculated intra-

Digitized by Google

TABLE No. IV.

TABLE SHOWING VIRULENCE OF THE TUBERCLE BACILLUS FROM VARIOUS SOURCES FOR RABBITS AND GUINEA-PIGS.

Culture.	Age in days.	Animals.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Date of Inoc.	Died.	Killed.	Length of Life.	Average.
J_6 Human	20	Guinea-Pig	610 grms.	370 grms.	240 grms. loss	July 16	Sept. 24		70 days	
,, ,,	20	,, ,,	514 ,,	320 ,,	194 ,, ,,	,, 16	Oct. 1		77 ,,	$79\frac{2}{3}$ days
,, ,,	20	,, ,,	434 ,,	310 ,,	124 ,, ,,	,, 16	,, 16		92 ,,	
,, ,,	20	Rabbit	920 ,,	1450 ,,	530 ,, gain	,, 16		Dec. 19	156 ,,	
,, ,,	20	,,	855 ,,	1470 ,,	615 ,, ,,	,, 16		,, 19	156 ,,	156 ,,
I_6 ,,	32	Guinea-Pig	695 ,,	510 ,,	185 ,, loss	,, 16	Aug. 4		19 ,,	
,, ,,	32	,, ,,	700 ,,	420 ,,	280 ,, ,,	,, 16	Sept. 12		58 ,,	$54\frac{1}{3}$,,
,, ,,	32	,, ,,	454 ,,	360 ,,	94 ,, ,,	,, 16	Oct. 10		86 ,,	
,, ,,	32	Rabbit	900 ,,	1850 ,,	950 ,, gain	,, 16		,, 19	156 ,,	
U_3 ,,	24	Guinea-Pig	390 ,,	210 ,,	180 ,, loss	Jan. 14	Feb. 20		37 ,,	
,, ,,	24	,, ,,	360 ,,	290 ,,	70 ,, ,,	,, 14	,, 21		38 ,,	43 ,,
,, ,,	24	,, ,,	330 ,,	265 ,,	65 ,, ,,	,, 14	March 9		54 ,,	
,, ,,	24	Rabbit	1710 ,,	2040 ,,	330 ,, gain	,, 14		June 15	152 ,,	
,, ,,	24	,,	1490 ,,	2130 ,,	640 ,, ,,	,, 14		,, 15	152 ,,	152 ,,
W_3 ,,	19	Guinea-Pig	745 ,,							
,, ,,	19	,, ,,	730 ,,							
,, ,,	19	,, ,,	700 ,,							
,, ,,	19	Rabbit	1470 ,,							
,, ,,	19	,,	1750 ,,							
F_6 Bovine	23	Guinea-Pig	404 ,,	320 ,,	84 ,, loss	,, 19	Sept. 7		50 days	
,, ,,	23	,, ,,	480 ,,	320 ,,	160 ,, ,,	,, 19	Aug. 16		28 ,,	$39\frac{1}{3}$ days
,, ,,	23	,, ,,	546 ,,	340 ,,	206 ,, ,,	,, 19	,, 28		40 ,,	
,, ,,	23	Rabbit	2350 ,,	1380 ,,	970 ,, ,,	,, 19	Nov. 23		127 ,,	
,, ,,	23	,,	910 ,,	670 ,,	240 ,, ,,	,, 19	,, 22		126 ,,	$126\frac{1}{2}$,,
Q_4 ,,	35	Guinea-Pig	380 ,,	270 ,,	110 ,, ,,	,, 19	Aug. 4		16 ,,	
,, ,,	35	,, ,,	510 ,,	420 ,,	90 ,, ,,	,, 19	,, 12		24 ,,	$24\frac{2}{3}$,,
,, ,,	35	,, ,,	800 ,,	560 ,,	240 ,, ,,	,, 19	,, 22		34 ,,	
,, ,,	35	Rabbit	1580 ,,	980 ,,	600 ,, ,,	,, 19	Oct. 13		86 ,,	
,, ,,	35	,,	895 ,,	875 ,,	20 ,, ,,	,, 19	Sept. 30		73 ,,	$79\frac{1}{2}$,,
QQ_5 ,,	25	Guinea-Pig	560 ,,	380 ,,	180 ,, ,,	Aug. 4	Aug. 22		18 ,,	
,, ,,	25	,, ,,	510 ,,	370 ,,	140 ,, ,,	,, 4	Sept. 3		30 ,,	24 ,,
,, ,,	25	Rabbit	1930 ,,	1500 ,,	430 ,, ,,	,, 4	Oct. 17		74 ,,	
,, ,,	25	,, *	1340 ,,	1080 ,,	260 ,, ,,	,, 4	Aug. 11		7 ,,	,,
T_3 ,,	35	Guinea-Pig	390 ,,	290 ,,	100 ,, ,,	Sept. 11	Oct. 21		40 ,,	
,, ,,	35	,, ,,	420 ,,	265 ,,	155 ,, ,,	,, 11	,, 10		29 ,,	$36\frac{1}{3}$
,, ,,	35	,, ,,	455 ,,	340 ,,	115 ,, ,,	,, 11	,, 21		40 ,,	
,, ,,	35	Rabbit	1210 ,,	1090 ,,	120 ,, ,,	,, 11	,, 18		37 ,,	
,, ,,	35	,,	1090 ,,	860 ,,	230 ,, ,,	,, 11	Nov. 12		62 ,,	$49\frac{1}{2}$,,

* This Rabbit died after seven days, from other causes, and is not included in the averages.

Digitized by Google

peritoneally. This was chloroformed on September 12 and cultures made. On October 11, no growth having taken place, the blocks of tissue were transferred to fresh serum, and stirred. On December 4 growth was first observed, and on December 15 sub-cultures were made. Growth has been rapid and abundant.

Culture W (human).—Obtained from a child nine months of age, of scrofulous parentage. The cervical glands on both sides were enlarged. Autopsy. A double broncho-pneumonia was found. One small tubercular nodule was found on surface of left lung, several at apex of right lung. Bronchial glands not enlarged. Tonsils normal in size, but glands on each side of pharynx extending in front of and behind sterno-mastoid muscle were enlarged, tuberculous, and several contained creamy pus. Three mesenteric glands were enlarged and cheesy: an ulcer was found in jejunum and another at ileo-cecal valve. Peyer's patches and the lymphatic glands about cecum were enlarged. Spleen and liver contained tubercles. The mesenteric glands were employed for the isolation of the culture.

This case was considered by Dr. Hand as being probably of tonsillar or pharyngeal origin.

Guinea-pigs were inoculated intra-peritoneally on October 5, 1900. From one which died on January 4, 1901, a second was inoculated intra-peritoneally. This was killed March 4, and cultures made. Growth was first observed on April 12, and sub-cultures made the following day.

Culture BB (human).—Obtained from a child seventeen months of age. Death was due to tubercular meningitis. Autopsy. Lungs normal, except posterior and part of right upper lobe, which was consolidated, red on section, and had excess of connective tissue. Bronchial and mediastinal glands not enlarged. Spleen, liver, and kidneys show pearly tubercles. Two feet from lower end of ileum is an ulcer 1 by 2 cm., the long diameter transverse to axis of gut. Nodules are seen on peritoneal surface. Four small ulcers, two above and two below, are found in ileum. Mesenteric glands enlarged, cheesy, and some purulent. Meninges show yellow tubercles most marked along longitudinal fissure, in choroid plexus, and over cerebellum. The mesenteric glands were used in obtaining the culture.

This case is considered by Dr. Hand the clearest one of primary intestinal tuberculosis ever seen by him.

Guinea-pigs were inoculated intra-peritoneally with an emulsion made from the mesenteric glands on March 9, 1901. One was chloroformed on April 1, and cultures made. Scanty growth on one tube was found June 8, and sub-cultures made.

The results given in Table IV. show for all the bovine cultures a greater virulence than for any of the human cultures. The average life of the guinea-pigs inoculated with human bacilli was more than twice that of those inoculated with bovine. All the rabbits which received human cultures gained in weight, and had finally to be killed, while for the bovine they died, and presented post mortem extensive lesions, the lungs and kidneys being the chief seats of the disease in all animals.

Digitized by Google

Part II.—Testing the Pathogenic Power of Tuberculous Material of Human and Bovine Origin.

In addition to the study of a number of pure cultures obtained from man and cattle, we have examined the pathogenic power of tuberculous material from the two sources also, making a series of inoculations which are as nearly parallel as possible. The results of these tests are shown in Table V and VI. The plan was to inoculate a series of animals with tuberculous tissues rubbed into a smooth suspension, and at the same time to inoculate a number of guinea-pigs with the same, which on their death would furnish material for another series of inoculations parallel with those made directly from the human and bovine tissues. It was believed that the material from guinea-pigs would more nearly represent pure cultures of the respective organisms than would be obtained from the tuberculous organs of man and cattle, and that the two sets of animals would lend themselves to an interesting comparison, besides fulfilling the primary object of comparing the virulence of human and bovine material.

The plan was carried out for the bovine material, but failed for the human, as all the guinea-pigs died within forty-eight hours after inoculation. The second series of animals were inoculated with human material from another source, and obtained at a later date. The results, while not entirely comparable, are nevertheless interesting and valuable.

Objections to the use of tuberculous material.—There are several disadvantages in using tuberculous material which were not overlooked in planning this work. (1) It is impossible to give accurate doses, for although our suspensions may be made of an equal opacity, and counts of equal amounts show approximately the same number of bacilli, there is no way of determining what proportion of these bacilli are capable of multiplication. (2) Material from man almost always contains other bacteria. Our knowledge of mixed infections is too scant to enable us to estimate the part played by these other species in determining the result; whether they inhibit or aid the action of the tubercle bacillus. The same is true of bovine material, although to a less extent, the tuberculous foci being more apt to remain enclosed. On the other hand, infection under natural conditions is always through such mixed material, and this method of inoculation approaches in some degree the conditions we meet in practice.

Source of bovine tuberculous material.—The bovine tuberculous material was obtained from a cow six years old, which had shown symptoms of tuberculosis for a long time, and was finally condemned and killed. She showed, post-mortem, a generalised involvement of the thoracic cavity with some invasion of the abdominal organs. In the lungs but little tissue approaching normal was found, and the pleuræ were studded with small nodules. Many of the tuberculous areas in the lungs had undergone caseation. The mediastinal glands were enormously enlarged, and caseous. Scattered over the peritoneum were many nodules. The liver was considerably involved, and the portal and mesenteric glands were enlarged and caseous. Other

Digitized by Google

abdominal organs showed no macroscopic lesions. Nodules were taken from the pericardium, mediastinal and mesenteric glands, lung and pleura. After thorough trituration in a mortar, each part separately, they were mixed, sterile water added, and the whole strained through cheese cloth. With this material a series of animals was inoculated, and at the same time a number of guinea-pigs. These all contracted tuberculosis, four dying on the fifteenth day. Portions of their organs were prepared in the same way, and used for the inoculation of a second series of animals, as seen in Table V.

Sources of human tuberculous material.—This material was obtained from three bodies, the lungs only being used. Two of the three were cases of well-marked acute miliary tuberculosis, the other a case of chronic phthisis, all adults. The material was prepared for injection in the same way as described for the bovine, the suspension being made as nearly of the same thickness as possible. The first series of animals was inoculated with the tissues from one of the cases of acute miliary tuberculosis, on August 27. A number of guinea-pigs were also inoculated with the object of having their tissues for a second series of inoculations, as with the bovine material, but they all died of septicemia within forty-eight hours, preventing the execution of this plan. With the exception of the pigs, and the calves inoculated with sputum, the other animals were inoculated from the two cases mentioned above.

Method of inoculation.—In all the animals the injection was made into the lung, the field of puncture being shaven, washed with soap and water, next with alcohol and ether, and lastly with a $\frac{1}{1000}$ solution of bi-chloride of mercury. The site of puncture was for the horses between the eighth and ninth ribs; for the pigs, sheep and dogs, between the sixth and seventh ribs, and for the cats between the fifth and sixth ribs.

Condensed Post-mortem Notes.

Horses. Bovine material. Two animals.—One died after fifty-four days, with loss of 195 pounds; the other was killed after sixty-six days, having lost 60 pounds. In both the disease was mainly in thoracic cavity. In the one inoculated with material direct from cow, both lungs were involved throughout. In the other the tuberculous process was confined to lung into which injection was made, and was marked only near site of inoculation.

Horses. Human material. Two animals.—Both had to be killed. One was entirely normal, not even the point of inoculation being discernible. In the other, the tuberculous process was confined to an area 5 cm. in diameter, with the point of inoculation as a centre.

Pigs. Bovine material. Four animals. All died, with an average length of life of fifty and three-quarter days. The two which received the bovine material direct lived sixty-eight and a half days, as against thirty-three days for those inoculated from the guinea-pigs. In all the lungs were the chief seat of the disease, all showing an acute miliary tuberculosis of both organs. In the two inoculated directly from the cow, the abdominal cavity was invaded to a limited extent, seen only in the mesenteric glands.

Digitized by Google

TABLE No. V.

TABLE SHOWING RESULT OF INOCULATION OF ANIMALS WITH TUBERCULOUS MATERIAL FROM BOVINE AND HUMAN SOURCES.

Material.	Reference Number.	Animals.	Date of Inoc.	Method of Inoc.	Dose in c. c.	Initial Weight.	Weight at Death.	Loss or Gain in Weight.	Died.	Killed.	Length of Life.	Average.
Bovine	9838	Horse	July 14	Intra-Pulmonary	10	1205 lbs.	1010 lbs.	195 lbs. loss	Sept. 6		54 days	60 days
,,	9840 *	,,	,, 30	,,	10	1075 ,,	1015 ,,	60 ,, ,,		Nov. 14, 1899	66 ,,	
,,	9701	Pig	,, 14	,,	4	130 ,,	78 ,,	52 ,, ,,	Aug. 23		40 ,,	
,,	9702	,,	,, 14	,,	4	131 ,,	103 ,,	28 ,, ,,	Oct. 19		97 ,,	
,,	9708 *	,,	,, 30	,,	5	94 ,,	86 ,,	8 ,, ,,	Sept. 12		44 ,,	50¾ ,,
,,	9706 *	,,	,, 30	,,	5	85 ,,	72 ,,	13 ,, ,,	Aug. 21		22 ,,	
,,	9697	Sheep	,, 14	,,	5	66 ,,	49 ,,	17 ,, ,,	,, 27		44 ,,	
,,	9696 *	,,	,, 30	,,	5	71 ,,	49 ,,	22 ,, ,,	,, 31		32 ,,	38 ,,
,,	9841	Dog	,, 15	,,	5	34½ ,,	24 ,,	10½ ,, ,,	Jan. 15, 1899		184 ,,	
,,	8010 *	,,	,, 30	,,	5	28 ,,				Mar. 20, 1899	245 ,,	214½ ,,
,,	8089	Cat	,, 18	,,	2·5	2508 grms.	1695 grms.	813 grms. ,,	Aug. 18		31 ,,	
,,	8001 *	,,	,, 30	,,	5	1150 ,,	1043 ,,	107 ,, ,,	,, 19		20 ,,	25½ ,,
Human	8008	Horse	Aug. 27	,,	10	1205 lbs.	1225 lbs.	20 lbs. gain		,, 14, 1899	199 ,,	
,,	8088	,,	Sept. 27	,,	10	1200 ,,	1370 ,,	170 ,, ,,		,, 14, 1899	168 ,,	183½ ,,
,,	9704	Pig	Aug. 27	,,	5	80 ,,	100 ,,	20 ,, ,,	Dec. 9		104 ,,	
,,	9842	,,	,, 27	,,	5	91 ,,	91 ,,	0 ,, ,,	Nov. 8		73 ,,	
,,	9844 †	,,	Nov. 9	,,	5	70 ,,	71 ,,	1 ,, ,,	Dec. 14		35 ,,	
,,	8095 ‡	,,	Dec. 14	,,	5		193 ,,			,, 21, 1899	97 ,,	58⅓ ,,
,,	9705 †	,,	Nov. 9	,,	5	87 ,,	107 ,,	20 ,, ,,	,, 7		28 ,,	
,,	8094 §	,,	Dec. 7	,,	5				,, 20		13 ,,	
,,	9698	Sheep	Aug. 27	,,	5	70 ,,	74 ,,	4 ,, ,,		,, 17, 1899	202 ,,	
,,	9699	,,	,, 27	,,	5	58 ,,	79 ,,	21 ,, ,,		,, 17, 1899	202 ,,	202 ,,
,,	12179	Dog	Sept. 8	,,	5	55 ,,	28 ,,	27 ,, loss		,, 25, 1899	229 ,,	
,,	12180	,,	Aug. 27	,,	5	29 ,,	32 ,,	3 ,, gain	Oct. 29		68 ,,	146 ,,
,,	10023	Cat	,, 27	,,	2·5	2190 grms.			Aug. 29			
,,	10060	,,	Sept. 8	,,	5				Mar. 20, 1899		193 ,,	
,, Sputum	5984	Calf	July 29	Intra-Peritoneally	10	108 ,,	258 ,,	150 ,, ,,		Jan. 14, 1899	169 ,,	
,, ,,	8499	,,	,, 29	,,	10	131 ,,	265 ,,	134 ,, ,,		,, 14, 1899	169 ,,	
,, ,,	8049	,,	May 16	,,	10		340 ,,			Aug. 1, 1898	77 ,,	
,, ,,	8050	,,	,, 16	,,	10		190 ,,			,, 1, 1898	77 ,,	
,, ,,	8074	,,	Oct. 18	Fed 11 times	30 to 60	224 ,,	385 ,,	161 ,, ,,		Feb. 18, 1899	123 ,,	132½ ,,
,, ,,	8096	,,	,, 18	,, ,, ,,	30 to 60	230 ,,	350 ,,	120 ,, ,,		,, 18, 1899	123 ,,	
,,	9843 ‖	,,	Aug. 2	Intra-Peritoneally	20	108 ,,				Jan. 10, 1899	161 ,,	
,,	9846 ¶	,,	,, 2	,,	20	132 ,,				,, 10, 1899	161 ,,	

* These animals were inoculated with the tissues of guinea-pigs which had died of general tuberculosis due to inoculation with material from a tuberculous cow. This material was the same as that used for the inoculation of the other animals.

† Inoculated from tissues of pig 9842. ‡ Inoculated from tissues of pig 9844. § Inoculated from tissues of pig 9705.

‖ Inoculated from tissues of calf 8049. ¶ Inoculated from tissues of calf 8050.

Digitized by Google

Pigs. Human material. Six animals.—Five died, and one was killed. The average length of life of the two inoculated directly from man was eighty-eight and a half days; for the three inoculated with tissues of other pigs which died, the average was twenty-two days. In the two first the lungs were invaded throughout with miliary tubercles, and one showed areas of pneumonic character. In neither was the abdominal cavity involved.

Of the four pigs inoculated with material from the other pigs which had succumbed to inoculation with human tissues, three presented a diffuse tubercular pneumonia, rapidly fatal, one living only thirteen days. The remaining animal was normal, with the exception of the right lung and pleura. Both visceral and parietal pleuræ were largely covered with nodules 2 mm. to 12 mm. in diameter, many of them caseous. The lung was dense throughout, and contained many caseous areas, most marked near the surface.

Sheep. Bovine material. Two animals.—Both died, the one inoculated directly with bovine material in forty-four days, the other in thirty-two days. Both were considerably emaciated, and in both the disease was confined almost entirely to the thoracic cavity, the spleen being the only abdominal organ involved, and this only slightly so. In both the lungs showed hepatization, more marked in the one inoculated with the tissue of the guinea-pig.

Sheep. Human material. Two animals.—Both were killed 202 days after inoculation. One was entirely normal, with the exception of an area in the right lung 5 cm. in diameter at the point of inoculation, in the centre of which was a caseous abscess, the pus containing many tubercle bacilli. The second animal was normal throughout.

Dogs. Bovine material. Two animals.—The dog which received the bovine material direct became markedly ill, much emaciated, and died after 184 days. The pleural surfaces of both lungs were covered with minute nodules, and were almost completely consolidated. In the right lung at point of inoculation was a small abscess 2·5 cm. in diameter.

The dog inoculated with tissues of guinea-pig showed little effect from it, and was killed after 245 days. The only lesion found was an abscess cavity 12 mm. in diameter at point of inoculation in right lung, scrapings from the wall of which contained many tubercle bacilli.

Dogs. Human material. Two animals.—One killed after 229 days. The only evidence of disease was the presence of numerous minute nodules on the pleural surfaces of both lungs, and a few on the pericardium. The lungs were normal otherwise.

The second dog began to cough after six weeks, lost flesh rapidly, and died on the sixty-third day. Both pleuræ contained 6 ounces of sanguinolent pus. The lungs were the seat of an extensive miliary tuberculosis, and at point of inoculation in right lung was an abscess cavity 7 cm. long by 2·5 mm. in diameter. The abdominal organs were not involved.

Cats. Bovine material. Two animals.—Both became rapidly ill, one dying in twenty and the other in thirty-one days. In both the lungs were the seat of an acute miliary tuberculosis. In the one which received the bovine material direct the spleen contained a number of cheesy areas. In the other

Digitized by Google

the mesenteric glands were enlarged and cheesy. The more rapid progress of the disease in the latter was probably due to its smaller size and the larger dose.

Cats. Human material. Two animals.—One cat died after two days, and has not, therefore, been included in the averages. The second was killed after 193 days, and found to be normal throughout. Not even the point of inoculation could be determined.

Summary.—Twelve animals were inoculated with bovine material. Ten of these died, while two survived—the horse and the dog, which received the tissues of the guinea-pigs. Fourteen animals were inoculated with the human material, of which eight died and six were killed. For most of the animals the tuberculous material direct from the cow seemed to be the more virulent. The sheep and pigs were exceptions to this—the pigs notably so, those receiving the tissues of guinea-pigs dying in thirty-three days, as against sixty-eight and a half days for the others. The averages of life after inoculation given in the table, as well as the greater mortality, show strikingly the increased potency of the bovine material over that from man.

This difference is further brought out by the extent and character of the lesions produced by the one and the other, as shown in the post-mortem notes, and in Table VI.

Infection of calves with human tuberculous sputum.—The results of this experiment are included in Table V., the details, which are of considerable interest, being supplied here.

Calf 5984, age five weeks, weight 108 pounds, was inoculated intra-peritoneally with 10 c.c. of sputum from an advanced case of tuberculosis at the University Hospital, on July 29, 1898. The patient from whom the sputum was obtained was a young adult who had been sick about a year and a half, and had been expectorating freely for one year. A cavity had been observed in the left lung about eight months before the sputum was obtained. Death occurred soon after. Beyond some slight elevation of temperature this calf showed no effect whatever from the inoculation. On November 3, 1898, it was tested with tuberculin, but gave no reaction. When it was killed on January 14, 1899, it weighed 256 pounds, an increase of 150 pounds. It was in good condition and showed no tuberculous lesions in any part of the body. The site of inoculation could not be determined.

Calf 8499, age five weeks, weight 131 pounds, was inoculated on July 29, 1898, with 10 c.c. of sputum of a patient at the University Hospital. This patient was an adult male; a miner by occupation. He had been under treatment for nine months. At this period the apices of both lungs were consolidated. He had had a cough for two years, lost flesh, and had one severe hæmorrhage. He expectorated a large amount of muco-purulent sputum. This calf showed an elevation of temperature which continued almost without intermission for three months, reaching as high as 105·8° F. in August and September. On November 3, 1898, it was tested with tuberculin and gave a reaction. It was killed on January 9, 1899, weighing 265 pounds, an increase in weight of 134 pounds. It was in fair condition. There was a nodule in

Digitized by Google

TABLE No. VI.

TABLE SHOWING COMPARATIVE EXTENT OF LESIONS PRODUCED BY INOCULATION OF HUMAN AND BOVINE MATERIAL.

—	Bovine Material Direct.	Bovine Material passed through Guinea-Pigs.	Human Material Direct.	Human Material passed through other animals.
Horses	Extensive disease of thoracic cavity.	Extensive involvement of lungs.	One horse limited involvement of one lung. The other was normal throughout.	
Pigs	Extensive disease of thoracic cavity.	Extensive disease of thoracic cavity.	Extensive Disease of thoracic cavity.	Rapidly fatal very acute process confined mainly to lungs. Slower process in one, confined to lung and pleura of one side.
Sheep	Extensive disease of thoracic cavity.	Extensive disease of thoracic cavity.	Limited involvement of one lung and pleura of same side in one animal. The other normal throughout.	
Dogs	Extensive disease of thoracic cavity.	Limited involvement of one lung.	Limited involvement of pleura and pericardium in one animal. In the other extensive disease of thoracic cavity.	
Cats	Extensive disease of thoracic cavity and spleen.	Extensive disease of thoracic cavity and mesenteric glands.	Normal throughout.	

Digitized by Google

the peritoneum at the site of inoculation. In the omentum were numerous calcareous nodules, the size of a hazel nut, and one softened nodule the size of a hen's egg, which contained an inspissated purulent material, and five smaller ones of the same character. The lungs, liver, and spleen were normal, but on the pleural surface of the diaphram there was a deposit of fibrin which contained several well-defined nodules. On the abdominal surface of the diaphragm was a small amount of grape formation.

Calf 8049, age about four weeks, was inoculated on May 16, 1898, intra-peritoneally with 10 c.c. of sputum from an advanced case of pulmonary tuberculosis at the University Hospital. The sputum contained many bacilli. Beyond a slight cough, which was noticed in July, it showed no symptoms of discomfort, and grew rapidly. It was tested with tuberculin on July 30 and gave good reaction. It was killed on August 1, weight 340 pounds, condition good; large amount of fat. On the surface of both lungs and on the pleural surface of the diphragm was a fibrinous deposit. In the cervical lobe of the right lung a few small nodules were found on section. The mediastinal and bronchial glands were somewhat enlarged. The omentum contained a few nodules, and one, about one inch in diameter, which had undergone softening, the contents being rich in tubercle bacilli. The spleen showed a number of nodules. About one-half of the mesenteric glands were enlarged and showed cheesy degeneration. An emulsion from the centre of the softened nodule in the omentum and from portions of the spleen was made, and 20 c.c. injected intra-peritoneally into calf 9843, four weeks old, weight 108 pounds, on August 2. Beyond a slight cough, noticed on October 10, the animal showed no ill-effects from inoculation. It was killed on January 10, 1899; condition good. The omentum was adherent to the peritoneum at the point of inoculation, and contained some ten nodules about the size of a pea. In every other respect the animal was entirely normal.

Calf 8050, age four weeks, was inoculated on May 16, 1898, with sputum from an early case of pulmonary tuberculosis at the University Hospital. The sputum contained a large number of tubercle bacilli. Soon after inoculation the temperature of the calf rose and continued high, with some remissions, until it was killed. Its appearance was bad, the coat dry and rough, the respiration rapid. It was tested with tuberculin, but the temperature was too high for results. It was killed on August 1, weighing 190 pounds. On the surface of both lungs there was a slight deposit of fibrine, and on section a number of hæmorrhagic areas were observed in both. The mediastinal and bronchial glands were enlarged and congested. The abdominal cavity contained about 12 ounces of bloody serum. The peritoneum was thickly studded over its entire surface with nodules from 1 mm. to 12 mm. in diameter, fibrous in character. In many places these nodules had massed together, forming tumours, some 5 cm. in diameter, which were dense and fibrous. The spleen contained many nodules both on the surface and throughout its substance. The whole omentum was thickly studded with nodules from 2 mm. to 12 mm. in diameter, and besides which there were three large masses, dense and fibrous in character, two of which were 15 cm. long by

Digitized by Google

7 cm. wide, and 12 mm. thick; and the third 7 cm. long by 6 cm. wide, by 4 cm. thick. The abdominal surface of the diaphragm was thickly studded with nodules, fibrous in character. The mesentery was thickened and contained many nodules of small size. The appearance was that of a typical case of grape or pearl disease. The mesenteric and mediastinal glands were enlarged and somewhat caseous. Twenty c.c. of an emulsion made from these glands, which contained a large number of tubercle baccilli, was injected on August 2 into the peritoneal cavity of calf 9846, four weeks old, weight 132 pounds. The animal showed no ill-effects whatever from inoculation, and was killed on January 10, 1899. A careful post-mortem examination showed it to be normal in all respects.

Summary.—Four calves of nearly the same age received intra-peritoneally 10 c.c. of human tuberculous sputum from different sources, but in all cases containing a large number of tubercle bacilli.

One showed no ill-effect from the injection except a slight rise of temperature, and when killed the autopsy was entirely negative.

Of the other three, two had persistent high temperature following the injection, but only one showed marked illness otherwise. Post-mortem examination proved that all had become infected with tuberculosis, the lesions in two being quite extensive. From each of these two a second calf was inoculated intra-peritoneally with an emulsion made from well-developed nodules. In both cases the emulsion was rich in tubercle bacilli, and a large dose —20 c.c.—was injected. The result was absolutely negative in one animal, and practically so in the other.

Since both calves received a much larger number of tubercle bacilli in the emulsion than those injected with the sputum, we are led to conclude that the result in the latter was due to a mixed infection which operated to the advantage of the tubercle bacillus.

The attempt to infect calves with human sputum by the digestive tract failed wholly. Two young calves (Table V., Nos. 8074 and 8096) were given from 30 to 60 c.c. of sputum, containing many tubercle bacilli, on eleven days. Some disturbance of digestion resulted at the time, but when killed no trace of tuberculosis was detected.*

Indications for further investigation.—In the further elucidation of this question, cultures should be isolated from as many cases of primary intestinal tuberculosis as possible, and especially those cases in which there is reason to suspect infection by meat or milk. Having obtained the cultures, it may well be asked whether or not we are in a position to determine positively the origin of the offending organisms. Are the differences which have been noted in culture, morphology and virulence, sufficiently marked and persistent to make differentiation possible? This I doubt. In my judgment much more work must be done before we will be able to determine the origin of a given culture of the tubercle bacillus by examination of it in cultures. At present we know practically nothing of the influence of the human body on the

* This experiment was conducted by Dr. W. G. Shaw, to whom all the credit is due.

Digitized by Google

tubercle bacillus, nor what changes may be induced in its morphology, cultural peculiarities and virulence by residence in the human tissues, nor what length of time is necessary to induce such changes, if induced at all. In my studies I have, as shown in Table III., recovered the tubercle bacillus, both human and bovine, from horses, dogs, swine, goats, and, in one instance, had the rare, if not unique opportunity, of recovering the bovine organism from man after accidental inoculation. These recovered cultures have all been carefully compared with the originals, and while some differences have been observed, and noted in the remarks following the table, with possibly one exception, they have not been of a marked or distinguishing character. In the case of the man, the bacillus remained in his tissues from January 1 to February 27, fifty-eight days. The recovered culture was practically identical with the original. This is, however, a shorter time by a great deal than elapses between intestinal infection in children through food and their death, and does not enable us to draw conclusions as to these cases. Like the culture recovered from animals, it indicates that the tubercle bacillus is quite tenacious of its characteristics as a rule. We have noted constantly that the bacilli found in scrapings from the various organs of the animals inoculated with bovine cultures have been long and beaded, though the culture used was of the short and unbeaded type. The recovered cultures, however, on blood serum have always resembled the original. In a series of examinations of material coughed up by cows, I failed always to find bacilli of what has been described as the bovine type. In fact, the longest tubercle bacilli I have ever observed, except in old cultures, were seen in some specimens of this material. The virulence of this material was greater considerably than human sputum ordinarily is, though the comparison was not made with accuracy.

Retention of characteristics in culture.— On blood serum with 5 per cent. of glycerine, the tubercle bacillus from bovine sources will, as a rule, retain its morphological and cultural characteristics for a long time. Culture H has now been grown for two years (July 6, 1901), and is of the sixteenth generation. Beyond an increased vegetation, no marked change can be detected in it. With a single exception (culture F) the same may be said of all the cultures isolated. This culture was isolated with some difficulty, and so scant was the growth for six generations that I was several times on the point of abandoning it. Only in the sixth generation was growth enough obtained for experimental inoculation. Considerably more growth took place in the seventh, and the eighth grew luxuriantly, since which all sub-cultures have been abundant. From the eighth generation on serum cultures were made on 5 per cent. glycerine agar, and an abundant growth obtained on this medium in the first transfer. Coincident with this increase of vegetative power came a marked change in the morphology. From being short, thick, and staining evenly, it is now long, more slender than in the early growths, and shows marked beading. In other words, from being a typical "bovine" culture, it has, during the past ten months, so changed that it can now pass as a typical "human" culture. It will be observed, by reference to Table IV.,

Digitized by Google

that its pathogenic power for guinea-pigs and rabbits was not as great as is usually found in bovine cultures.

Culture in collodion sacs.—The method of culture in the body of living animals, which has been productive of such brilliant results in the hands of the French, offers much assistance in the solution of the problem before us. It has enabled Nocard* to demonstrate the possibility of so modifying the mammalian tubercle bacillus that it becomes like the avian organism in culture and pathogenicity. We are now attempting to modify two feebly virulent human cultures by residence in the peritoneal cavity of cattle.

Culture M was kept for seven months in the abdominal cavity of a heifer, enclosed in collodion sacs, two sacs being used. On removing the sacs but little multiplication of the bacilli was noted. Cultures were recovered directly from the sacs. In the first transfer the cultures grew more rapidly and abundantly than the original culture, but the morphology of the individual bacilli cannot be said to have changed to a noticeable degree. The virulence was not increased. Rabbits were not killed by sub-cutaneous inoculation. Guinea-pigs died after an average of 42⅓ days. A calf inoculated intra-peritoneally showed no ill effect, and gave no reaction to tuberculin after seven weeks.

Culture Nasua Narica (Coati) of Theo. Smith, enclosed in collodion sacs, was kept in the peritoneal cavity of a yearling heifer for eleven months. On removal there was considerable increase in the growth, especially in one sac. Cultures were recovered from the sacs, and showed some marked changes, especially in manner of growth. On both blood serum and glycerine agar the growth was most rapid, at least twice as rapid as usual for this culture. On serum it covered the surface in seven to ten days, as a thick, white, moist layer, almost cottony near the water at the bottom. On glycerine agar the growth was more dry and wrinkled, but very rapid. The bacilli stain more evenly, and are, on the whole, shorter, though long forms are seen. Guinea-pigs and rabbits inoculated sub-cutaneously are still living after four months. A calf which received 4 c.c. of a milky suspension in the jugular vein reacted to tuberculin after ten weeks.

The experiment is being continued, the culture recovered from the capsule having been placed again in the peritoneal cavity of another calf.

No conclusions can be drawn as yet, though the indications are that both cultures have become less strictly parasitic rather than more so by the procedure.

Interpretation of results.—Accepting it as proven that the bovine tubercle bacillus has, as a rule, considerably greater pathogenic power than the human bacillus for a large majority of experimental animals, how should we interpret this in regard to man? Is it fair to conclude that this increase of virulence will hold good for man also? Until the contrary is proven, or until good reason for believing the contrary is shown, it is in my judgment right that this conclusion be held, at least as a working hypothesis. I am aware of the

* 'Annales de l'Institut Pasteur,' Vol. XII., No. 9, September, 1898.

Digitized by Google

objections to this view that will be raised by some, and acknowledge freely that it cannot be accepted as conclusive. Virulence is, no doubt, a factor which, is relative to the subject, and exaltation of virulence for one species does not necessarily prove an increased virulence for other species. Indeed, the reverse is true in some instances.* However, it cannot be denied, as a general rule, that when the virulence of a pathogenic organism is increased for one animal, it is increased for all that are naturally susceptible to its action.

The tubercle bacillus is unique in the extent of its pathogenic activity, both by direct experimental inoculation as well as by infection under what may be considered more or less natural conditions. The list of animals in which tuberculosis has been observed in parks and zoological gardens is appalling, the discoveries of Dubard † and others showing that not even the cold-blooded animals are exempt from this universal scourge. While it may be said of the tubercle bacillus that in cultures in the laboratory it is unusually tenacious of its characteristics, it is certain that in Nature it has a wide range of adaptability as a pathogenic agent. Hence, for the tubercle bacillus, perhaps more than for any other known microbe, we are justified in believing that an exaltation of virulence for practically all experimental animals will hold good in the case of man also.

The question can be determined definitely only by direct inoculation of man. To do this experimentally is, of course, impossible, consequently we are forced to rely for evidence of this nature on those accidental cases which occur from time to time. It has been my fortune to have three such cases come under my observation, in each of which the infecting organism was known positively to be of bovine origin.‡ Similar cases have been reported by Tscherning § and Pfeiffer,‖ the latter ending in general infection and death. To these may be added two cases observed by Dr. M. B. Hartzell,¶ of the University of Pennsylvania, though in both absolute proof of the bovine origin of the offending organism is lacking. Both occurred in healthy men employed by one of our large American railways to clean and repair cars used in the transportation of cattle. In both a well-developed tuberculosis of the skin followed slight wounds of the back of the hand inflicted by broken timbers. In one case the local disease was soon cured, and no further trouble resulted. The other, however, ended fatally after about a year, through the infection becoming generalised, with involvement of the lungs. This patient was a robust man, forty-four years old, weighing 175 pounds, with good family and personal histories. Dr. Hartzell felt able to exclude with reasonable certainty any other source of infection.

Cases such as these permit us to deny with authority the claim which has

* The streptococcus is said to become increased in virulence for mice by successive passages through these animals, but less virulent for rabbits.

† 'La Revue de la Tuberculose,' April, 1898.

‡ 'Philadelphia Medical Journal,' July 21, 1900.

§ 'Congrès pour l'étude de la Tuberculose,' 1st Session, 1888.

‖ 'Zeitschrift f. Hygiene,' Bd. III., 1898.

¶ 'Journal American Medical Association,' April 16, 1898.

Digitized by Google

been made by certain persons that by long residence in animals of the bovine species the tubercle bacillus becomes so changed as to render it incapable of successful residence in the tissues of man. In all of these instances the bovine bacillus grew and multiplied under conditions known to be most unfavourable to it, with the production of characteristic lesions, and in two of the seven cases gained access to the internal organs, causing death, a result which is unusual when the local lesion is due to infection from human sources. While the number of cases is too small to enable us to draw sweeping conclusions, the indications are that by this mode of inoculation the pathogenic power of the bovine bacillus is at least as great as that possessed by its human congener.

Infection through food.—It will not be necessary here to review at length the reported instances of infection through the digestive tract, following the use of food products from tuberculous cattle. Most of these of necessity lack precision, and are not absolutely demonstrative, though some of them have, as Nocard has said, "almost the value of an experiment."

The well-known observations of Stang,* Demme,* Gosse,* and Ollivier† leave little doubt of the power of the bovine tubercle bacillus to infect man through the digestive tract.

Statistical evidence.—The evidence derived from the statistics of tuberculosis on this point is purely circumstantial, yet of such strength as to be most convincing. The compilations of Dr. Tatham bring out most strikingly the fact that in early life some potent factor is at work in causing tuberculosis. In the Harben Lectures for 1898, Sir Richard Thorne-Thorne speaks of this as follows: "So, also, if you will compare the rates in Tables A, B, and C (pages 5, 6, and 7), and contrast the reduction of 27·9 per cent. which has taken place, under five years of age, during the last forty-five years in all forms of tubercular disease, and that of 66 per cent. in phthisis, with the corresponding one from tabes mesenterica, which only reached 3·0 per cent., you will see that, in considering the latter cause of death, we are dealing with a totally different state of affairs.

"The matter, too, assumes a still more serious aspect if we limit ourselves to the first year of life, when milk is most largely used as food; for then we find that the reduction in the rate of death from the various forms of tuberculosis, which reduction has been going on at 'all ages' for about half a century, not only disappears, but is actually transformed into a large increase, reaching no less than 27·7 per cent. This in itself is grave enough, but its significance is still further emphasized when we remember what are the circumstances under which this increase in the rate of death from tabes mesenterica has gone on synchronously with a decrease in that from other forms of tuberculosis."

Evidence of a similar nature is given by Dr. Still,‡ of the Great Ormond

* "Les Tuberculoses Animales." Nocard.

† 'Paris Academy of Medicine.' 'La Semaine Médicale,' February 25, 1892.

‡ 'British Medical Journal,' August 19. 1899, and 'Journal of Comparative Pathology and Therapeutics,' Vol. XII., Part 4.

Digitized by Google

Street Hospital for Children, in his analysis of 769 consecutive autopsies of children under twelve years of age, 269 of which showed tuberculous lesions. Of these, 117, or 43·5 per cent., were in children under two years old, while in the first three years of life 56·5 per cent., or more than half of the total number, occurred. From his study of the lesions in these cases, Dr. Still believes that in 153, or 56·8 per cent., the respiratory tract was the channel of infection, while in 63, or 23·4 per cent., the alimentary canal was responsible, the remaining fifty-three cases being uncertain or otherwise accounted for. Accepting these figures as given, they indicate strongly that milk, the most largely used food, has a considerable part in the spread of tuberculosis, and justify the conclusion quoted from a report made to the Council of the British Medical Association, that "the mortality from tuberculosis in early childhood is not decreasing as it is at other ages in the United Kingdom, and the opinion that this great prevalence of the disease in childhood is due to infection through the alimentary canal by milk from tuberculous cows appears to be well founded."

From Germany come further confirmatory facts. Widerhofer gives an analysis of 418 cases of tuberculosis in children, showing among them 101 with involvement of the intestine. Of these, 43, or 42·5 per cent., were between the ages of two and five years, the period of life when cows' milk forms a large part of the food for children.

Conclusions.—In view of the foregoing experiments, and of the evidence quoted, it seems justifiable to conclude:—

1. That the tubercle bacillus from bovine sources has in culture fairly constant and persistent peculiarities of growth and morphology, by which it may tentatively be differentiated from that ordinarily found in man.

2. That cultures from the two sources differ markedly in pathogenic power, affording further means of differentiation, the bovine bacillus being very much more active than the human for all species of experimental animals tested, with the possible exception of swine, which are highly susceptible to both.

3. That tuberculous material from cattle and from man corresponds closely in comparative pathogenic power to pure cultures of the tubercle bacillus from the two sources, for all animals tested.

4. That it is a fair assumption from the evidence at hand, and in the absence of evidence to the contrary, that the bovine tubercle bacillus has a high degree of pathogenic power for man also, which is especially manifest in the early years of life.

The CHAIRMAN: This is a most interesting paper, and should be discussed to-morrow morning with the other papers. Those who have waited until the end of the meeting to-day will agree that they have not wasted their time, but that they have listened to one of the most important papers which has been given to this Congress.

Digitized by Google

Dr. BENDA made the following explanatory statement :—

Meine Herren,—Ich muss noch einige Augenblicke Ihre Aufmerksamkeit erbitten. Ich sehe, dass ich für den nächsten Britischen Tuberculose-Congress noch sehr viel mehr englisch lernen muss als bisher. Ich habe leider Dr. McWeeney vollständig missverstanden und muss mich entschuldigen, dass ich in meinem Schlusswort Ausführungen von ihm widerlegt habe, die er nicht gemacht hat. Ich glaube, dass das am besten aus der Welt geschafft wird, wenn ich Sie bitte, das zu streichen, was ich *gegen* ihn gesagt habe, und ihm zu danken für sein Interesse an meinen Untersuchungen. (Applause.)

Auch ich hoffe ebenso wie Dr. McWeeney, dass wenn die Aufmerksamkeit auf die Gefässtuberculose gerichtet wird, sie reichlicher gefunden werden wird. Ich habe die erste 1894 gefunden; und ich bin erstaunt, wie viele es giebt.

Professor MCWEENEY: I have to thank Professor Benda for his very kind words. He did misunderstand me, but he has now put that right.

(The meeting then adjourned.)

Digitized by Google

EXCRETION TUBERCULOSIS OF THE KIDNEY.

By HUGH WALSHAM, M.A., M.D. Cantab, F.R.C.P. Lond.,

Assistant Physician, formerly Pathologist, to the City of London Hospital for Diseases of the Chest, Victoria Park.

During the last two years (I began the work in July, 1898) I have been making some observations on the position or lie of tubercle bacilli in the medulla of the kidney in cases of disseminated miliary tuberculosis, and, as a result of these observations, I have ventured to describe these medullary tubercles as tubercles of excretion.

It will be well, I think, at the outset of this paper to define as clearly as possible what I mean by the term "excretion tuberculosis." Briefly, I mean this—that tubercle bacilli, having gained entrance to the urine tubes by a process of excretion through the glomeruli, become arrested at some point in the tube, most often in the medulla of the kidney, and there give rise to secondary tubercular foci or tubercles of excretion.

I have examined thirteen cases of disseminated miliary tuberculosis with reference to this point. The results obtained may not seem commensurate to the time taken up with the investigation, but material had to be waited for and a great number of microscopical sections had to be rejected, the absence of bacilli or the advanced stage of caseation of the tubercles making it impossible to draw any conclusion as to their origin. Indeed, as I proceeded with the investigation, I found that all tubercles that had advanced to the stage of giant-cell formation had to be rejected. The action of the bacilli appears to be weakest in the vicinity of the giant cell; in fact, the giant cell is an indication that the tubercle is passing into a chronic condition; moreover, the bacilli found in the neighbourhood of the giant cells are usually scanty in number (excepting those found in various stages of disintegration within the protoplasm of the cell), according to Metschnikoff* and Stschastny,† on account of the phagocytic action of the giant cell on the bacilli. I shall say no more on the subject of giant-cell formation in renal tuberculosis, with the exception that they are probably not formed from the renal epithelium, as maintained by Arnold.‡ The functions of the renal epithelium on the one hand, and the protoplasmic body called the giant cell on the other, are so diverse that the formation of the latter from the former is very hard to conceive. In all my cases serial sections were made in order to determine

* *Metschnikoff.*—'Virchow Archiv.' Bd. 113.
† *Stschastny.*—'Virchow Archiv.' Bd. 115.
‡ *Arnold.*—'Virchow Archiv.' Bd. 83.

Digitized by Google

the position of the tubercle bacilli as accurately as possible, tracing them section by section. The kidneys were hardened in Müller's fluid and alcohol. The sections were stained in carbol-fuchsin, decolourised in a 25 per cent. solution of sulphuric acid and counter-stained in Methylin blue.

The microscope used in this investigation was Zeiss Stand V^a, with Abbe condenser, plane mirror, and open diaphragm. The objectives were Zeiss A and D and Leitz $\frac{1}{12}$ oil immersion. The eye-pieces Zeiss 2 and 4. In using the term "high power," I mean in every case the $\frac{1}{12}$ oil immersion of Leitz, and Zeiss ocular, either No. 2 or 4.

If we look in the more modern text-books of pathology we find little or nothing stated as to the exact position occupied by the bacilli in miliary tuberculosis of the kidney. Professor Ziegler,* in his handbook (last German edition, 1898), says: "Die Tuberkulose der Nieren entsteht meistens durch hämatogene Infection, doch kann auch eine in den Harn ableitenden Wegen localisirte Tuberkulose auf die Niere Übergreifen. Die Miliartuberkulose ist meist Theilerscheinung einer über einen mehr oder minder grossen Theil der Organe verbreiteten Tuberkeleruption. Da, wo die Bacillen hingelangen, erscheinen zunächst kleine, helle, graue verwaschene Flecken. Weiterhin entwickeln sich graue Knötchen, die später weiss werden und häufig von einem hämorrhagischen Hof ungeben sind. Die grauweisse Verfärbung ist theils durch eine Wucherung des Bindegewebes, theils durch eine trübe Schwellung und Nekrose des Epithels bedingt. Innerhalb der Tuberkel gehen die einzelnen Nierenbestandtheile zu Grunde. Die Zahl der Tuberkel, die in einer Niere sich entwickeln, ist bald bedeutend, bald gering. Zuweilen ist die Eruption auf das Gebiet eines Astes der Nierenarterie beschränkt."

Professor Coats,† in the last edition (1900) of his Manual of Pathology, says:—

"In acute general tuberculosis there are numerous tubercles in the kidneys, mostly in the cortex. They are usually elongated in shape and visible to the naked eye as small pale areas. Under the microscope they are found to be caseous in their central parts, while peripherally their cells infiltrate between the tubules."

Professor Hamilton ‡ is the only British pathologist who mentions the position occupied by the bacilli. His description is so full and accurate that, at the risk of being tedious, I have quoted it at length. He says:—

"This (acute miliary tubercle of the kidney) is always associated with tubercle in other organs—that is to say, it is part of a general eruption. The nodules vary in size from a millet-seed up to a large pea, the most of them being of the former dimension. They are most abundant in the cortex, either immediately adjacent to the boundary zone or at the extreme surface. They are exposed, consequently, on removing the capsule. When of any size they are always yellow in the centre, owing to early caseation. They have a very sharp boundary margin, and vary in shape according to the locality. Thus

* *Ziegler.*—"Lehrbuch der Pathologischen Anatomie," 1898. Zweiter Band.

† *Coats.*—"Manual of Pathology," 1900.

‡ *Hamilton.*—"Text-book of Pathology," Vol. ii. Pt. i.

Digitized by Google

Digitized by Google

Plate I.

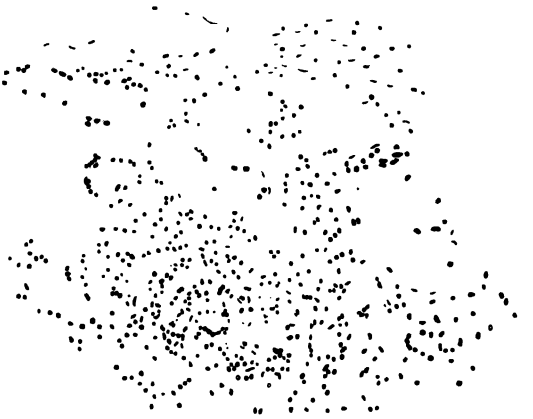


Fig. 1.

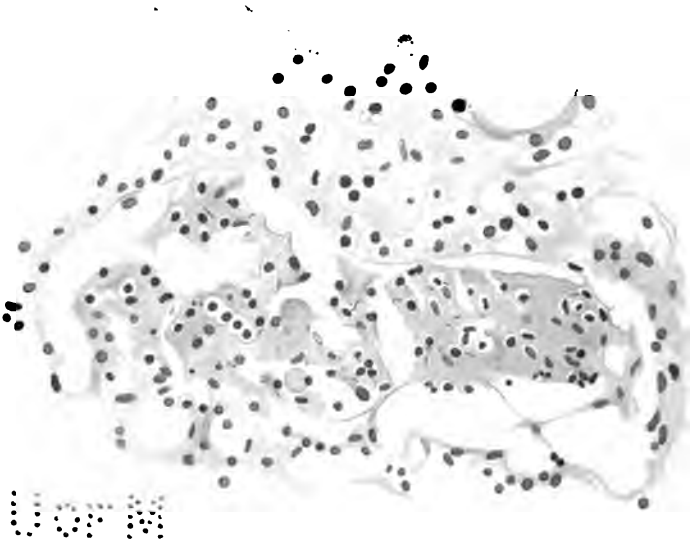


Fig. 2.

To face page 585

Digitized by Google

towards the surface of the cortex they are usually wedge-shaped, further in they are rounded, *while in the medulla they are long fusiform bodies.*

"Microscopically they are usually found to be composed of masses of round cells without much reticular network; giant cells rapidly make their appearance; and the centre caseates so soon as the nodule becomes a naked-eye object. Tubercle bacilli are usually scanty. *When present they are found within the afferent arteries and glomeruli, sometimes in the interstitial tissue, and occasionally in the tubes.* As the tumour continues to grow, the blood supply is gradually cut off from it, so that in time not a particle of injection can be driven into its interior, even although the vessels of the kidney generally are quite pervious.

"The nodule appears to rise within a minute blood-vessel. This is what might be naturally expected, seeing that the bacillus is conveyed by the blood. The blood-vessel is rapidly destroyed, and the cellular accumulation, of which the tubercle at first consists, spreads thence between the tubes, surrounds them, and pushes them aside. Those which happen to be engulfed in the cellular mass are strangled and destroyed by it. This variety of tubercle has little inclination to soften, either because the disease proves fatal before time is afforded for this happening, or because the nodules have an inherent tendency to pass into a fibrous state and to contract. When a nodule reaches a large size a little cavity may be noticed within its substance, but it seems doubtful, to say the least of it, if this variety of tubercle ever terminates in true renal phthisis."

I will now give the cases.

Case I.—A man, aged forty-eight, died in the City of London Hospital for Diseases of the Chest, of morbus cordis. Numerous miliary tubercles were found scattered in the peritoneum and kidneys. Lungs were emphysematous, but free from tubercle. The tubercles in the kidneys were found scattered in the cortex and medulla, each being surrounded by a zone of congestion. The post-mortem was made by Dr. J. J. Perkins, assistant physician to St. Thomas's Hospital, but at that time pathologist to the City of London Hospital for Diseases of the Chest.

The kidney was examined by serial sections.

In one of these sections (see Plate 1, Fig. 1) a small beginning tubercular nodule was selected at the junction of the cortex and medulla. Under a low power of the microscope the central part of the tubercle is seen to be occupied by a urine tube, the basement membrane of which is intact. The epithelium lining the tube is dull and cloudy, retracted from the basement membrane and partly broken up. Caseation has only just begun. The tube, owing to the alteration of its epithelial lining, stands out in sharp contrast to the surrounding normal urine tubes. Numerous lymphoid cells are seen infiltrating the surrounding tissue.

Under a high power (see Plate 1, Fig. 2), Leitz $\frac{1}{12}$ oil immersion, many tubercle bacilli are seen lying among the broken up and cloudy epithelial cells of the tube, but none are to be discovered among the surrounding lymphoid cells. This tubercular nodule was traced through eight serial sections, and in all of

Digitized by Google

them the bacilli occupied the same position (varying, of course, in numbers) in the tube, with none in the surrounding tissue.

I next examined a very small commencing tubercle in the medulla of the same section (see Plate 2, Fig. 1). The urine tubes are in transverse section. Under a low power, in the centre of the tubercle is seen a small red spot or ring, surrounded with numerous lymphoid cells. Under a high power (see Plate 2, Fig. 2) this red spot is seen to consist of masses of tubercle bacilli, lying amongst the broken up epithelium of a urine tube, the urine tubes and interstitial tissue in the neighbourhood being quite free from bacilli, but showing evidence of commencing lymphoid infiltration. This tubercle was traced through eight serial sections. The bacilli, as in the former case, were confined to the urine tube, none being present in the interstitial tissue.

The inference from the above case is, I think, conclusive. In each tubercle we have a urine tube as a centre, filled with tubercle bacilli, with surrounding lymphoid cells. It appears to me clear that the bacilli have become arrested in the tube and have there given rise to a tubercular focus. In fact, they are excretion tubercles.

Case II.—The next case was that of a man aged twenty-eight. Miliary tuberculosis of lungs, spleen, kidneys.

There were numerous miliary tubercles scattered through cortex and medulla.

The kidney, as in the former case, was examined by serial sections.

A tubercular nodule was discovered in the lower part of the cortex. The tubercle, in this instance, was further advanced in caseation than in the former. A urine tube is seen at the periphery of the nodule. The epithelium has stained badly owing to the caseation. The lymphoid accumulation is well seen. The basement membrane of the tube is still to be made out, notwithstanding the caseation of the epithelium. Under a high power seven tubercle bacilli are seen within the tube, while others are scattered among the broken up and caseous epithelium of the neighbouring urine tubes. The tubercle is an elongated one, as they usually are in the lower cortex and medulla of the organ. This tubercular nodule was traced through twelve serial sections, the bacilli occupying the same position in each.

Case III.—Disseminated miliary tuberculosis of lungs, spleen, liver, and kidney in a girl aged sixteen.

In examining the sections of the medulla of this kidney a large collecting tube was discovered filled with the broken up detritus of its lining epithelium. Scattered in this granular mass were numerous tubercle bacilli; the small tubes in the neighbourhood are quite normal. In this instance the bacilli were in a process of expulsion through the tube. They had not given rise to any tubercular formation in the surrounding tissue. This tube was traced through thirteen serial sections, all sections containing bacilli. Again, from another part of the same section (see Plate 3, Fig. 2) two urine tubes were discovered with the same granular and broken up epithelium; a few tubercle bacilli were scattered in the larger tube, but only one was discovered in the smaller. The surrounding insterstitial tissue is quite free from bacilli or any

Digitized by Google

Plate II.

Fig. 1.

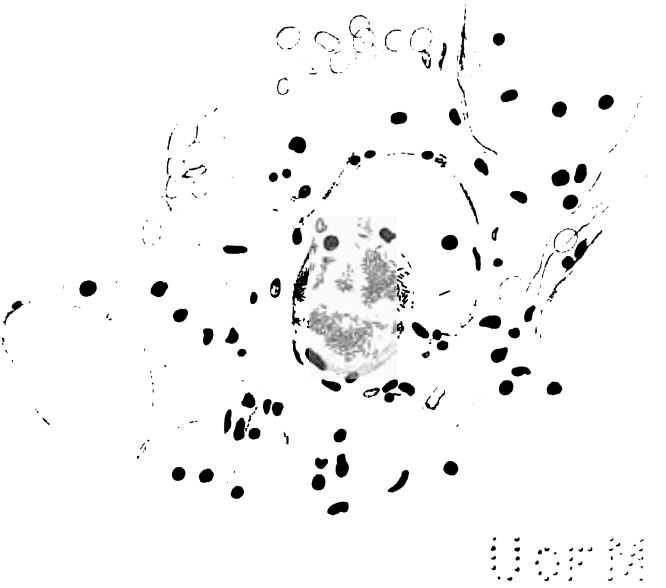


Fig. 2.

To face page 586.

Digitized by Google

Digitized by Google

tuberculous change. In another section of the same kidney a tube is seen in transverse section of the medulla, the epithelial lining for the most part has been desquamated; six tubercle bacilli are seen in the desquamated epithelium. There is also a large cell whose nuclei are undergoing a process of division, and is no doubt on its way to the formation of a giant cell. Serial sections were made in this case also.

Case IV.—A man, aged twenty, died in the City of London Hospital for Diseases of the Chest, of acute miliary tubercle. Many grey granulations were found scattered through the cortex and medulla of the kidney. A small beginning tubercle was found in the medulla. It was elongated in shape, as tubercles usually are in this position. The basement membrane of the tubules could still be traced through the tuberculous nodule. The epithelium of the tubes was broken up in parts, mingling with the lymphoid cells. Numerous tubercle bacilli were found in the epithelium of the tubules. The tubercle was traced through six serial sections, the tubercle bacilli occupying the same position in each section.

It is not my intention in this paper to enter into the very wide question of the excretion of micro-organisms other than the tubercle bacillus by the kidney, but there can be, I think, no doubt that other pathogenic bacilli and cocci are also excreted by the organ. Dr. Horton Smith,* in the Goulstonian Lectures, delivered before the Royal College of Physicians of London, on the typhoid bacillus and typhoid fever, in discussing the pathology of the typhoid bacillus in the urine, writes as follows: "How is the bacilluria produced, and on what does it depend? The simplest explanation which suggests itself—namely, that we have to do with a mere filtration of the bacilli from the blood—may be dismissed at once. The extreme difficulty in finding the bacilli in the blood during life would point to its improbability. Moreover, I have on four occasions examined the blood bacteriologically, at a time when the urine was swarming with bacilli, and on all occasions I failed to find the typhoid bacilli therein. A second suggestion is that the condition arises from suppuration set up in the kidney by the typhoid bacillus, accompanied by the secondary passage of the bacilli and pus into the urine. Such cases do undoubtedly occur, one being recently recorded by Flexner,† but they are extremely rare. Thus, out of 289 post-mortem examinations on patients who had died from typhoid fever at St. Bartholomew's Hospital during the last thirty years, in one case only were abscesses found in the kidneys. This rare condition, therefore, could hardly explain the very frequent presence of the bacilluria.

"We are driven, then, to our third possibility, to offer an adequate explanation of the condition. According to this view, which I believe to be correct, the whole would depend on a rapid growth in the urine itself within the bladder of the bacillus, a stray micro-organism having found its way there from the blood, doubtless after its passage through the kidney."

---

* *Horton Smith.*—'Goulstonian Lecture,' 1900, and 'Medico-Chirurgical Transactions,' Vol. lxxx.

† *Flexner.*—'Journal of Pathology and Bacteriology,' 1895, Vol. ii.

Digitized by Google

Dr. Horton Smith has then to assume that a stray typhoid bacillus has to find its way through the kidney, but how can this take place except by a process of excretion? He says that the extreme difficulty of finding typhoid bacilli in the blood during life would point to its improbability, but we must remember that the same difficulty is experienced in the discovery of tubercle bacilli in the blood during life in cases of disseminated miliary tuberculosis, although after death many miliary tubercles are found in the kidney, proving conclusively that the bacilli must have been present in the blood during life. I do not for one minute dispute Dr. Horton Smith's contention that the typhoid bacilli multiply in the bladder, but I maintain that they must first be excreted by the kidney.

One cannot help regretting that Dr. Horton Smith did not make an histological examination of the kidney in his cases, with the intent to discover the presence of the typhoid bacillus in the glomeruli or tubules of the organ. It is interesting to note that Dr. Mayer* has found small foci of suppuration in tuberculous kidneys in a case of chronic pulmonary tuberculosis with excavation, resembling those described by Drs. Horton Smith and Flexner in the typhoid kidney. This is not surprising when we remember that many cases of so-called chronic pulmonary tuberculosis are in reality cases of mixed infection. I have not met with this condition myself, owing, no doubt, to my cases being of the miliary variety. As I said above, it is not my intention in this paper to discuss generally the excretion of micro-organisms by the kidney, but I should like to call attention to a section which shows a portion of the cortex of the kidney in a case of septicæmia. Two of the urine tubes will be seen stuffed with micrococci, while the surrounding tissue is free from them, but is infiltrated with pus cells. There can be, I think, no doubt that these cocci were in a process of excretion by the kidney at the time of the death of the patient.

The results obtained by the experimental production of renal tuberculosis are of very great interest as bearing on this question—although experimental observations are not necessarily homogeneous with those of morbid anatomy. Professor Baumgarten† was the first observer to produce experimentally tubercle of the kidney. He says: "Nächst den Lungen sind es die Nieren, welche in unseren Versuchen am frühesten und reichlichsten von metastatischer Tuberkelentwicklung ergriffen werden. Wie bereits oben erwähnt, bleiben die in die Nieren eindringenden Tuberkelbacillen nachweislich zunächst theils in Schlingen der Glomeruli, theils in Epithelien der gewundenen Harncanälchen, in welche letztere sie wahrscheinlich von den anliegenden Capillargefässen aus, möglicherweise auch mittelst des Harnstroms (dem sie Seitens der Glomeruli beigemengt werden können hineingelangen, haften."

The much more recent experimental work of MM. Kostenisch and Volkow‡ does not help us much on account of their method of inoculation.

---

* *Mayer.*—'Virchow Archiv.' Bd. 141.

† *Baumgarten.*—'Zeitschrift für klinische Medicin.' Bd. X. 1886.

‡ *Kostenisch and Volkow.*—'Archives de Médecine experimentale,' 4. 1892.

Digitized by Google

They injected tubercle bacilli directly into the kidney through the lumbar walls. But this method obviously complicates matters unnecessarily by adding to the tuberculous process a process of repair. Of great interest, however, is the experimental work of Borrel,* which he communicated to the Eleventh International Medical Congress held in Rome. Borrel first injected tubercle bacilli into the aorta in order to avoid the pulmonary filter, and describes this form of renal tuberculosis as "tuberculose primitive à localisation glomérulaire ou corticale prédominante"; and, secondly, he obtained on the twentieth day after injection into the venous system a granular tuberculosis, "Tuberculose granulique disséminée dans toutes les parties du rein à localisation périvasculaire prédominante." He goes on to say, "*Dans les deux cas, le processus tuberculeux est toujours interstitiel*, les seuls éléments actifs du tubercule sont des éléments lymphatiques—les éléments différenciés de l'organe, et l'épithélium en particulier, ne jouent aucun rôle dans la formation des tubercules." In the "tuberculose rénale primitive," he specially lays stress on the "localisation glomérulaire"—"je n'ai jamais constaté la présence de bacilles dans la substance des pyramides." Borrel maintains that his second form, "tuberculose granulique du rein," corresponds to "la granulie aiguë chez l'homme." This form "ne montre pas de localisation prédominante dans telle ou telle partie du rein." In the formation of the tubercle, "les tubes subissent un véritable processus d'atrophie et ne jouent aucun rôle actif." Their late appearance, their position around the vessels, and their relative poorness in bacilli, distinguish these granulations from the primary tubercle. According to Borrel, in this second form, after the primary spread of the bacilli by the blood-vessels has taken place, there is a secondary spread by the lymphatic of the kidney.

In Plate 4, fig. 3, Borrel shows cells with bacilli in a conduit "qui n'est sûrement pas un tube du rein, mais qui pourrait bien être un conduit lymphatique." I think, however, viewed in the light of my observations, this conduit is in reality a cross-section of a urine tube.

Of very great interest also, as bearing on this question, is the work of Wyssokowitsch.† He injected various micro-organisms, such as the bacillus pneumoniæ, the bacillus of enteric fever, into the blood of animals, such as rabbits, dogs, guinea-pigs, and then after varying intervals of time made plate cultivations of the urine sediment. He came to the conclusion that "Eine grössere Quantität der injicirten Bacterien findet sich nur dann im Harn der Versuchsthiere, wenn makroskopisch wahrnehmbare Blutextravasate oder Heerde in den Nieren vorhanden sind." Now, blood extravasations I have never observed in tubercular kidneys, but I am unable to say whether tubercle bacilli can be excreted by the kidney without foci of tubercle in the cortex, as all my cases contained tubercle in this region of the kidney. After the injection of the typhoid bacillus into the blood of dogs, Wyssokowitsch was unable to find the bacilli in the urine with plate cultivations. Dr. Horton

* *Borrel.*—'Annales de l'Institut Pasteur,' 8. 1894.

† *Wyssokowitsch.*—'Zeitschrift für Hygiene.'

Digitized by Google

Smith, however, has shown conclusively that they do occur in the urine in this disease in man. Wyssokowitsch does not appear to have made any observations with the bacillus of tubercle.

After the hitherto unrefuted results of Wyssokowitsch's trials we should not assume a simple excretion of the bacteria through the unaltered kidney. Konjajeff* has found small foci in the kidneys, mostly close under the capsule, and has demonstrated in them the presence of typhoid bacilli in culture and in sections. The author believes he is able to identify these foci as the incipient stages of the so-called acute lymphomas, which occur in the organs in typhoid fever and other infectious diseases. Konjajeff says: "Die Nierenlymphome haben in Betreff ihrer Beschaffenheit und Lage eine grosse Aehnlichkeit mit den Niereninfarkten. Letztere sind gar nicht selten, so fand Hoffmann dieselben auf 250 Typhusautopsieen 10 mal. Die kleinsten Infarkte haben oft die grösste Aehnlichkeit mit den lymphomatösen Knötchen, so dass nur die mikroskopische Untersuchung im Stand ist den Unterschied zu zeigen." Out of the seven cases described by Dr. Horton Smith one was fatal. The post-mortem note on the condition of the kidneys is as follows:—"The kidneys were large, swollen, and deeply coloured, and showed many small infarcts; some of these latter had broken down and had formed small abscesses." No microscopical examination is given, but one cannot help thinking that these so-called infarcts were Konjajeff's acute lymphomata.

From the above observations, then, we see that tubercle bacilli are either found in some part of the course of the uriniferous tubules surrounded by lymphoid accumulations, or they are found in the tubules far from any tuberculous focus and without any tuberculous change in the neighbourhood. From the surrounding tissue they cannot possibly have come; they must primarily have reached the tube either by a process of excretion through the glomeruli, or by rupture of a tuberculous caseous focus into the tube; but this latter method is very difficult of conception. If we look at Plate 3, Fig. 1, we see a glomerulus of the kidney with numerous bacilli in one-half of the capillary tube, without any tubercular change in the surrounding tissue. This is, I think, a glomerulus in the process of excreting the bacilli. Tubercle bacilli arriving by the renal artery become arrested either in the capillaries of the glomeruli or in a small twig of the renal artery, probably in consequence of the slowing of the circulation at these points. In the majority of cases the bacilli give rise to tubercular changes at these spots, but in a small number of cases the bacilli would appear to be excreted by the glomeruli into the uriniferous tubes.

But how is it that in some cases (I only found evidence of excretion in four cases out of thirteen examined with reference to this point) excretion of the bacilli takes place, but not in others? Macroscopically the picture is the same in both. Perhaps large caseous foci of tubercle hinder excretion. A caseous glomerulus is probably unable any longer to excrete, or we may suppose that the glomeruli undergo some alteration; the capillary wall

* *Konjajeff.*—'Centralblatt für Bacteriologie,' 5–6. 1889.

Digitized by Google

Plate III.

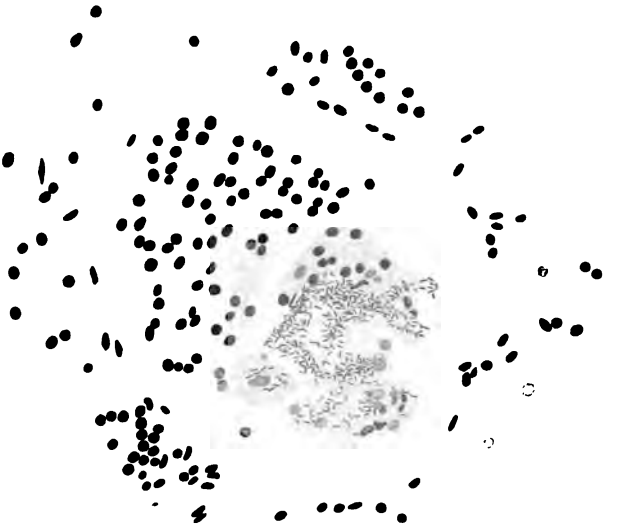


Fig. 1.

Fig. 2.

To face page 590.

Digitized by Google

Digitized by Google

undergoing some change under the influence of the tubercular virus, which allows the passage of the bacilli into the urine tubes. If, on the one hand, as frequently happens, we find large foci of tubercle with wide-spread caseation, containing apparently normal urine tubes which, notwithstanding many tubercle bacilli in the surrounding tissue, contain none, we have a right to assume that the bacilli have been conveyed by the blood or lymph vessels; but if, on the other hand, we find tubercle bacilli in a urine tubule with little surrounding caseation, this condition, I think, points to the conclusion that the bacilli have not found entrance to the tubes from the surrounding tissue, but have produced the tuberculous nodule while in a process of excretion by the kidney.

To sum up, I have shown the presence of tubercle bacilli within the glomeruli of the kidney without any visible change in the vessels of the glomerulus, or in the surrounding tissues. Next I have proved the presence of the bacilli in the urine tubes. Then I have shown the bacilli in the tubes setting up tuberculous changes in the neighbourhood or tubercles of excretion, and finally, the presence of the bacilli in the urine of a patient who died of acute disseminated miliary tuberculosis.

I have here to thank Dr. Arthur Francis Voelcker, assistant physician and late pathologist to the Middlesex Hospital; Dr. J. J. Perkins, assistant physician and pathologist to St. Thomas's Hospital; and Dr. O. K. Williamson, assistant physician, late pathologist, to the City of London Hospital for Diseases of the Chest, for kindly supplying me with the kidneys. I have also to thank *the artist, Mr. John R. Ford*, for the great care he has taken in drawing and colouring the plates from the microscopical preparations.

Digitized by Google

FRIDAY, JULY 26th, 1901.

PROFESSOR SIMS WOODHEAD, F.R.S.E., *President of Section*, *IN THE CHAIR.*

The proceedings commenced at 9 A.M. with a demonstration of a series of lantern slides by Dr. WILLIAM BROWN, of Carlisle, on "Bovine Tuberculosis."

The PRESIDENT: I think we must thank Dr. Brown very heartily for bringing this demonstration before us, and we are only sorry that we could not give him more time to go more thoroughly into the question. We have, however, an immense amount of work to get through in this our last day. We cannot conclude without thanking Dr. Brown very heartily for bringing his beautiful specimens and slides before us. There are, perhaps, one or two points which will be raised in a future discussion, and any which are in your minds may be raised on Professor Bang's paper. Professor Bang's paper is on "Some Experiments on the Temperature necessary for killing Tubercle Bacilli in Milk." Yesterday we had a paper by Professor Ravenel which there was not time to discuss, and we thought we would take the discussion this morning. It will save time if we take remarks on papers after they have all been read.

Professor BANG read a paper entitled "Some Experiments on the Temperature necessary for killing Tubercle Bacilli in Milk":—

The question of the *thermal death-point of tubercle bacilli* seems not to be solved; at least you will find different opinions stated from well esteemed authors of the latest years. I therefore hope that some experiments shortly made by me and my assistant, Mr. Stribolt, regarding the temperature necessary to kill *tubercle bacilli in milk*, will have some interest.

Our experiments regard milk secreted from a tuberculous udder; the appearance of the milk was not much altered, a little thinner than natural, and flocky, so that the results obtained refer to the natural conditions in which tubercle bacilli are met with in milk.

In one series of experiments we treated the milk in a quantity of 25 cc. in large test tubes, which we put into a water bath of a constant temperature of 90° C. In the midst of the milk was applied a thermometer fixed in a cork, and as soon as the thermometer showed the degree of temperature which we wanted, the tube was removed and cooled in ice water. It took from 1 min. 20 secs. to 2 mins. 50 secs. to reach the various temperatures from the beginning temperature of 20°.

Of the heated milk we injected 10 cc. into the peritoneal cavity of

Digitized by Google

rabbits. The result was that eighteen rabbits which received the milk, heated to 75° (4 rabbits), 80° (4), 85° (6), 90° (4) remained healthy (or died after some months of intercurrent diseases), while two rabbits injected with the raw milk died after about two months highly tuberculous.

In this series of experiments we could not avoid the formation of a thin pellicle on the surface of the milk, which, after the experience of Professor Theobald Smith, is supposed to protect the bacilli included therein against the influence of the heat, nor could we avoid the formation on the surface of a layer of foam, which might possibly have the same effect. And we found, on the other hand, that the temperature of the milk was not equal through the whole mass of milk—it was, indeed, about 5° higher near the surface than near the bottom—when we did not shake the tube, which we wanted to avoid,

Date of Injection.	Quantity injected.	Temperature of the Milk.	Time of Exposure.	Rabbits.	Date when the animal died or was killed.	Result.
March 23 .	15 cc.	85° C.	Momentary	1	Killed July 8 .	Sound
—	—	—	1 min.	1	Killed July 8 .	Sound
—	—	80° C.	Mom.	1	Killed July 16 .	Sound
—	—	—	1 min.	1	Killed June 6 .	Sound
—	—	75° C.	Mom.	1	Killed July 16 .	Sound
—	—	—	1 min.	1	Killed June 6 .	Sound
—	—	70° C.	Mom.	1	Killed May 4 .	Sound
—	—	—	1 min.	1	Killed June 4 .	No tuberculosis. (Pneumonia)
—	—	65° C.	Mom.	1	Killed May 4 .	*Tuberculosis* in moderate degree. The rabbit was rather fat
—	—	—	1 min.	1	Died April 2 .	No manifest pathological alterations
—	10 cc.	—	5 mins.	2	Killed May 4 .	No tuberculosis. One of them had a large cheesy deposit (not tuberculosis) in the peritoneal cavity
—	—	—	10 mins.	2	(*a*) Died March 20	No tuberculosis. (Peritonitis)
					(*b*) Killed Mar. 20	Sound
—	—	—	15 mins.	2	Killed June 10 .	Both sound
—	15 cc.	60° C.	Mom.	1	Died May 2 . .	*Highly tuberculous*
—	—	—	1 min.	1	Killed May 4 .	*Highly tuberculous*
March 24 .	10 cc.	—	5 mins.	4	(*a*) Died April 1	Peritonitis. No tubercle bacilli to be found
					(*b*) Killed May 4	*Tuberculous*, rather highly
					(*c*) Killed May 2	*Tuberculous*, rather highly
					(*d*) Killed May 12	*Tuberculous*, rather highly
—	—	—	10 mins.	4	(*a*)&(*b*) K. May 12	Both *slightly tuberculous*
					(*c*) Died April 15	No tuberculosis. (Pneumonia)
					(*d*) Killed June 10	*Tuberculous*, rather highly
—	—	—	15 mins.	4	(*a*) Killed May 21	*Very few tubercles* in the omentum, a mesenteric gland and the lungs
					(*b*) Killed May 21	Sound
					(*c*) Died May 25	*Very slightly tuberculous* (as *a*). Died of broncho-pneumonia
					(*d*) Killed June 6	Sound

Digitized by Google

because we feared that particles of the milk hanging on the thermometer might be protected against the heat.

In order to avoid all these possible sources of error and to secure a thorough heating of all particles of the milk, we altered our method of heating in the way that we included the sample of milk in a thin metallic box of cylindrical form, closed with a metallic cover, in which the thermometer was fixed. The box contained 30 cc. of milk, and when we put the whole deeply into a great water bath of constant temperature, shaking it the whole time, we were sure that no particle of the milk could avoid the influence of the heat. In such a metal box the milk very soon, after about 1 minute, reaches the temperature of the surrounding water. In some cases we cooled the milk in ice water as soon as the temperature wanted was reached, in others we kept it at this temperature 1, 2, 5, 10, 15 mins.

Of tuberculous milk treated thus we injected into the peritoneal cavity of rabbits 10–15 cc.

The result will be seen by the table on previous page.

Feeding Experiments.

With the same tuberculous milk we fed, *March* 17, a number of young rabbits. Each rabbit got 30 cc. of the milk heated in the above-mentioned way in the metallic box and kept *two minutes* at the temperature whose influence we would examine.

The result will be seen on the following table :—

Date of Feeding.	Quantity Given.	Temperature.	Animals.	Date when the animal died or was killed.	Result.
March 17 .	30 cc.	85° C.	2 rabbits	Killed July 8 .	Both sound
—	—	80° C.	2 rabbits	(*a*) Died April 20	No tubercle
				(*b*) Killed July 8	Sound
—	—	75° C.	2 rabbits	(*a*) Died June 7	No tubercle
				(*b*) Killed July 8	Sound
—	—	70° C.	4 rabbits	(*a*) Died March 28	No tubercle. (Was bitten to death)
				(*b*) Died May 24	No tubercle
				(*c*&*d*) Killed July 8	Both sound
—	—	65° C.	2 rabbits	Killed June 16 .	Both sound
—	—	60° C.	2 rabbits	Killed June 6 .	Both sound
—	—	*Raw milk*	2 rabbits	Killed June 2 .	Both *tuberculous ulcers* in the intestines (on the Peyer's plaque in the ileum and in the end of caecum). *Tuberculosis* in the mesenteric glands, in the liver spleen, kidneys and lungs

It will be seen that although the heating of tuberculous milk to 60° *C. for fifteen minutes* is *not able to destroy all tubercle bacilli* so as to prohibit infection, if you inject them into the peritoneal cavity, the *heating to the same degree for*

Digitized by Google

two minutes is enough to weaken them so much that they cannot infect through the alimentary canal.

We also made some *feeding experiments with pigs.* Each pig got 150 cc. of the same tuberculous milk, heated in the metallic box and kept half a minute at the temperature, whose influence we would examine.

The result was the following :—

Date of Feeding.	Quantity Given.	Temperature.	Animals.	Date when the pig was killed.	Result.
March 21 .	150 cc.	80° C.	1 pig .	June 16 . . .	Sound.
—	—	75° C.	1 pig .	July 1 . . .	Sound.
—	—	70° C.	2 pigs .	July 9 . . .	Both sound.
—	—	60° C.	1 pig .	June 13 . . .	*Tuberculosis* in the lymphatic glands near the throat, in the mesenteric, mediastinal and bronchial glands, and some nodules in the lungs.
—	—	*Raw milk*	1 pig .	June 13 . . .	Sound.

This experience does not, of course, prove much regarding the influence of the heat, as the pig that drank the milk did not acquire tuberculosis. As Mr. Stribolt himself saw it drink the tuberculous milk, the experiment has some interest, inasmuch as it proves that a pig may resist once feeding with a small quantity of tuberculous milk.

As to the influence which the quantity consumed has on the development of the disease, I wish to mention here some *experiments with calves* which we made two years ago. On March 4th we gave each of five sound calves ($3\frac{1}{2}$ months old) 40 *cc. of milk from a tuberculous udder*, and six days later they got *the same dose.* When these calves were killed, three to nine months after, we found them all tuberculous, with deposits in the mesenteric glands, some also in the supra-pharyngeal glands, and some with tuberculous ulcerations in the intestines. One of them was much emaciated and had severe diarrhœa. It had enormous tuberculosis in the bowels and the mesenteric and supra-pharyngeal glands, and also some tuberculosis in the lungs and bronchial glands.*

It is thus proved that calves may sometimes grow tuberculous after feeding with a very small quantity of tuberculous milk.

Experiments with Tubercle Bacilli in Culture.

In order to complete our researches on the influence of the temperature on tuberculous bacilli, we made some experiments with pure cultures. In small test tubes with glycerine bouillon we sowed small particles of the thin pellicle which a young culture of tubercle bacilli forms. The cotton plug was pushed

* Three of these calves were, before the feeding (and one of them also after it), treated with large quantities (128 cc. each) of the new tuberculin (T.R.), but it had no influence at all. The most affected was one of the treated.

Digitized by Google

downward and a caoutchouc cork (perforated by a small glass pipe with cotton lock) put over it.

These test tubes we put deeply down into a water bath in order to acquire as much security as possible for the thorough heating of the thin pellicle of culture which swam on the surface of the bouillon. We knew from experience that it takes about 1½ minutes before the bouillon has the same temperature as the surrounding water, and therefore we, no doubt, are right in speaking of a momentary heating to a certain degree. When we removed the test tube from the water bath after 1½ minutes and put it into cold water, accordingly we speak of 5 minutes' heating when we removed the test tube after 6½ minutes, etc.

The result of our experience was that the culture grew as well as in the control tubes after exposure to a temperature of 50° C. for 15 minutes; a little more slowly after 50° C. for 20 minutes.

55° C. had no influence by momentary action; after 5 minutes the growth was retarded, after 10 minutes some tubes got sterile, and after 15 minutes only one single tube showed a trace of growth.

60° C. made most tubes sterile after momentary action; in a few tubes there came a slow growth.

60° C. *for* 5 *minutes* made all tubes sterile.

65° C. made most tubes sterile after momentary action; in some tubes there was a doubtful trace of growth.

65° C. *for* 5 *minutes* made all tubes sterile; and the same was the case when the temperature had acted for a longer time, and when we exposed the tubes to higher temperatures as 70°, 75°, 80°, and 85°—in all cases no trace of growth.

These experiments with pure culture of tubercle bacilli correspond, then, quite well with the milk experiments; the influence of the heat is only a little stronger on the cultures. Maybe the cultivated bacilli are a little weakened, maybe they would be able to grow in the peritoneal cavity of an animal even after having lost the power of multiplying in bouillon with glycerine.

Our experiments have given almost the same results as those published by Professor Theobald Smith two years ago (*Journal of Experimental Medicine*, 1899)—he found tubercle bacilli destroyed at 60° C. in 15 to 20 minutes, and the larger number in 5 to 10 minutes—but they differ from what has been found by many other researchers, and also by myself many years ago. I occupied myself much in 1884 and the following years with experiments with milk from tuberculous udders, and I came to the decision that, although the heating to 65° *C. for* 5 *minutes* in some cases made the milk innocuous, this was not always the case. The same *inconstancy* I found for the higher temperatures, as 70°, 75°, and 80°. But at 85° C. I constantly found myself able to kill tubercle bacilli in milk. By *feeding experiments* in rabbits I demonstrated the weakening influence of 60° C. and 65° C., in so far that milk heated to these temperatures only produced very slight tuberculosis in some individuals, and in some cases was quite innocuous, although the feeding was continued for three months. At that time I felt inclined to seek the reason of the inconstancy of the result by injections in variability of the tubercle bacilli (perhaps spores), but nowadays I think it more probable that it has depended upon faults in the

Digitized by Google

method of treating the milk. As this was done in open test tubes put into a water bath, the formation of a pellicle on the surface has not been avoided, nor the formation of a layer of foam on the surface. And as no doubt the test tube was shaken a little, particles of milk may have been deposited on the thermometer above the surface of the milk, and in this way have been protected against the heat. Then the inconstancy may in some cases have depended upon the condition of the milk, which sometimes was rather serous and disposed to coagulation, and in some cases also contained rather large flocks, in whose interior the bacilli may have been somewhat protected.

I suppose that some of these circumstances may explain the fact that many other investigators have had results which also differ from those of Mr. Theobald Smith and Mr. Stribolt, and mine.

Most closely corresponding to our results are those of Dr. De Manns (*Archiv für Hygiene*, 1893). He found the tubercle bacilli killed after 15 minutes' exposure to 65° C., after 10 minutes to 70° C., and after 5 minutes to 80° C. But he relates no experiments about the influence of these temperatures applied for a shorter time, and his researches consequently do not enlighten us on the thermal death-point of tubercle bacilli.

The same thing may be said of the researches of Yersin (*Annales de l'Institut Pasteur*, 1888), who found that 70° C. in 10 minutes was able to kill the bacilli, but he did not try if 70° C. could have done the same thing in a shorter time. Nor did he try the influence of 65° C., but for 60° C. in 10 minutes he found a weakening influence.

Very different from my results are those of Prof. Max Beck (*Vierteljahrsschrift für off. Gesundh.-pflege* 1900). He found that the simple *boiling up* was not sufficient to kill the bacilli in natural tuberculous milk, and that milk mixed with tubercle bacilli was not made innocuous by heating to 80° C. for half an hour, but his experiments seem to have been done by heating the milk in open vessels, and it is allowable to believe that the formation of a pellicle or of foam on the surface has been the reason of the diverse results.

In the large series of researches made by Prof. Woodhead in 1895 for the Royal Commission on Tuberculosis, it will be seen that so long as he worked with natural tuberculous milk, most of his results corresponded very well with mine, inasmuch as milk heated to 60° C. for 5 minutes did not produce tuberculosis in guinea-pigs by feeding, and such milk heated to 60° C. for 25 minutes did not make them tuberculous after injection into the peritoneal cavity, and the same was the case with milk heated to 65° C. for 2½ minutes. Only in one experiment did he find that after heating to 70° C. for 5 minutes the milk made one out of three guinea-pigs tuberculous on injection, and the same result was got with two guinea-pigs injected with milk heated to 70° C. for 11–12 minutes, while 5 guinea-pigs which got the milk heated for 9–10 minutes remained free.

With milk mixed with finely divided tuberculous organs, Prof. Woodhead had very inconstant results, but I think it is allowable to believe that in these cases some tubercle bacilli have been protected against the heat through coagulation—a circumstance to which he himself calls attention.

Digitized by Google

Finally I wish to point out that it would no doubt be wrong immediately to draw *consequences for practice* from these scientific researches. In a common pasteurising apparatus for milk, as it is used in the dairies, the milk passes so fast (in some of them in a little more than half a minute) that it does not quite correspond to what I call momentary treating in our metallic box, and in most of them the milk is very foamy, and we have found that *the foam which comes out of such apparatus is not at all as hot as the milk.* Then the milk may in some cases contain large flocculi or coagulating material. It would, therefore, I think, be unfortunate to diminish the degree of heating in the common dairies. I think it best to continue heating the skimmed milk before sending it back to 80° or 85° C., as we do in Denmark. Only at this temperature is it possible to control the heating by a simple colour-proof (method of Storch, *i.e.*, shaking the milk with some drops of para-phenylen diamine and hydrogen dioxide).

The PRESIDENT: We are very much obliged to Professor Bang for this paper, and not least for the outcome of the question. He shows that when carrying out scientific experiments it may be possible to do certain things, for then we have everything to favour us in the laboratory. But in actual practice there are factors in the experiments which render it necessary to go very much beyond what is necessary in a scientific experiment. That is a very important matter indeed.

I think we might now, with advantage, discuss Dr. Ravenel's paper, and also Professor Bang's paper. Those who have remarks to make on these two subjects will kindly bring them as much as possible together, and so condense the discussion.

Dr. CARLO RUATA, *University of Perugia*: Mr. President and Gentlemen, —I would like to make one remark on Dr. Ravenel's paper, on a point which impressed me very much with regard to tuberculosis due to infected milk. I show here a table of statistics. All the statistics published on the

1899.—DEATHS IN ENGLAND FROM—

Age.	Tabes Mes.	Tub. Mening.	Phthisis.
0- 1	3,208	1,820	431
1- 2	1,100	1,228	394
2- 3	366	712	221
3- 4	164	478	134
4- 5	106	372	131
5-10	325	917	576
10-15	186	353	1,011
15-20	166	176	3,185
20-25	133	102	4-784
25-35	190	140	9,955
35-45	98	44	6,718
45-55	41	15	3,478
55-65	19	4	1,357

Digitized by Google

point agree that where meat is used intestinal tuberculosis is produced. The last figures I have been able to procure are those published in England in 1899. In the first year a very high death-rate is shown as due to tabes mesenterica in the first twelve months of life. Then in the subsequent periods the figure diminishes very much, until, when we get to the fourth or fifth year of life, it has fallen from 1,100 (in the first year) to 106 cases. Those are the years of life when meat is not used as food. I do not set that up as a proof against all that has been done in laboratories, but it seems to me it ought to receive consideration. Yesterday I heard Dr. Ravenel's address, and I remember well what he wrote in the Arbor Lectures. He (Dr. Ravenel) said in his Arbor Lecture that the high death-rate of 3,208 during the first year of life is produced by infected milk. I said, "You may be right; it may be produced by infected milk, but you have also 1,820 deaths from tubercular meningitis during the first year, and I do not think that infected milk can produce tubercular meningitis." Not only that, but we have another fact, that the highest mortality is during the first six months. The statistics in England are only published for the first year, and for no shorter period; but in Italy we have a return made for every month of the year. We there find that the death-rate from tabes mesenterica is as high for the first few months as for the first year altogether. Knowing that tuberculous tabes mesenterica is so slow in its progress I cannot say that this high death-rate during the first month is produced by infected milk. The death-rate from tubercular meningitis is so high that I cannot say it is secondary to tabes mesenterica; both are primary. I ask you to note from these tables the course of these three kinds of disease. The figures for them are very high at the beginning, but they then decrease in different degrees. But in phthisis we have an increase after the first five years; the figures go up from 1,011 to 3,100, 4,000, 9,000. While the figures for the two other forms are going down those for phthisis are going up. I think that is due to the fact that here begins the influence of external causes—inhalation of the tubercle bacillus, etc. I say that these three are forms of the same disease, and if there is a great difference in tabes mesenterica, it is due to the fact that in the fœtus the intestines are much larger and more frequently attacked than the meninges. If anyone can give a better explanation I shall be very pleased to hear it. One important thing, it seems to me, is this: that although these facts may not be conclusive, they are borne out by all the statistics on the subject which are issued.

The PRESIDENT: We have but a very short time at our disposal. I shall ask those who have remarks to offer to make them as short as possible, as we must get through the work of the Section. We also want to give Professor Ravenel an opportunity of saying a few words in reply. I trust speakers will keep to the point, and make their remarks as concise as possible.

Dr. KOSSEL, *Berlin*: If I may be allowed to speak a few words on the subject I would refer to experiments that have been carried out in the German Imperial Health Office by Regierungsrath Dr. Tjaden. Dr. Tjaden and I have,

Digitized by Google

in the course of the last year, visited a number of large dairies, one of which had a supply of 12,000 to 15,000 litres of milk per day. In these dairies the milk before being centrifugated is heated in special heating apparatuses. After the heating the milk is centrifugated, and the cream is used for making butter, whereas the greater portion of the milk, deprived of its cream by the centrifugating, is sent back to the different farms and serves as food for calves and pigs. The four different milk-heating apparatuses that were used in these dairies were based on the principle of what we call in German the "Zwangsläufige Führung," *i.e.*, the milk after entering the apparatus is forced to flow over surfaces heated by steam, and is kept in constant movement by special *Rührwerke*. The time which elapses between entering and leaving the apparatus is rather short, according to the apparatus used, from one to two minutes, rarely more. The temperature desired may be easily regulated. Dr. Tjaden injected guinea-pigs with the milk which had passed the apparatus, whereas other animals were inoculated with the milk before it was heated.

Other experiments were made with heating in the laboratory the milk of animals affected with udder tuberculosis at different degrees, and cooling it down subsequently as quickly as possible, thus exposing the milk to the desired temperature only for a few seconds. Finally he tested the efficiency of the above apparatuses by sending through them cows' milk which was mixed with milk from the same infected cows, thus imitating the natural conditions, having assured himself that the milk used contained tubercle bacilli.

The results were the following:—

In the laboratory experiments the degree of temperature at which the tubercle bacilli were killed varied considerably according to the physical condition of the milk used. Sometimes the bacilli were killed only after heating the milk to as high a degree as 98° C., whereas in other instances milk lost its infective properties after being heated to much lower degrees for the same short time. He therefore thinks that in laboratory experiments the results of different experimenters, and even of the same observer, must always vary to a certain extent. The results of the experiments with the milk of the aforesaid dairies yielded much more satisfactory results. In not a single case was an animal injected with heated milk found to be tuberculous, whereas the control animals showed signs of tuberculosis. This result was obtained even when the temperature had not been higher than 85° C. So it seemed that in using the aforesaid apparatuses the temperature required was considerably lower than in the laboratory experiments. To be quite sure of this he repeated the experiments in the same apparatus, using, as aforesaid, milk that was mixed with milk from cows affected with tuberculosis of the udder. The results obtained were the same as those with milk from the dairies. It could be observed that heating the milk even to as low a degree as 85°, for the short time in question, was sufficient to kill the tubercle bacilli. Dr. Tjaden, from this observation, draws the conclusion that it would be sufficient to instruct the dairies to

Digitized by Google

heat the milk to at least 90° C., in an apparatus based on the aforesaid principles, as one then may be sure that the milk, in any case, has been heated sufficiently. One might postulate this the more, as heating to 90° C. seems not to interfere in any way with the subsequent preparations of dairy products, such as butter and cheese. The same effect of killing the tubercle bacilli can, of course, be obtained by heating the milk to a lower degree for a longer time, but the cost in the latter case will be more considerable, and this method can be used only in large cities where the milk is sold as such, and not used for the preparation of butter and cheese. Dr. Tjaden's experiments thus have proved that to-day we are able to heat large quantities of milk—in some of the apparatus 6,000 litres per hour—to a desired degree which is high enough to deprive the milk of its infective properties.

Professor McWEENEY: I will confine my remarks to one single part of the subject matter. In Ireland we have many institutions called piggeries, in which milk is worked up. The cream is made up into butter. As Medical Adviser to the Local Government Board in Ireland, I have been consulted on the subject of the temperature to which milk should be raised in order to destroy its potential infectivity. I was not aware at the time of the facts which have been brought forward during this discussion; and accordingly the recommendation which I thought it safe to make was that the milk should be heated to 75° C. for a period of fifteen minutes; and I considered that if at the same time the formation of a pellicle were prevented by the constant agitation of the volume of milk so treated, the infective properties of the milk would, in all probability, be destroyed. I refrained from making any experiments on the subject for the reason that I had no naturally tuberculous milk at my disposal, and I consider that experiments made should be upon naturally tuberculous milk. I think the clumping of the bacilli, and the difficulty of their regular diffusion through the mass of milk when an attempt is made to artificially infect milk with tubercle, prevents experiments based upon this approximating to those on naturally infected milk.

I wish to say one word about Dr. Ravenel's paper, not by way of discussion—because Dr. Ravenel raised such a large number of important points that it would be impossible to take them up seriatim—but to express my sense of indebtedness to Dr. Ravenel. I say his contribution to this discussion is of an epoch-making character.

Professor SHERIDAN DELÉPINE, *Manchester*: Mr. President and Gentlemen,—It was not my intention to speak here this morning, but happening to come here at a time when a subject at which I have worked for several years was being discussed, and hearing also of the opinions which have been freely expressed—namely, that it is possible to rely on the exposure of milk to a temperature of 85° C. for a limited number of minutes to destroy the virulence of the bacillus in milk—I felt it almost a duty to express my dissent from those who, like Professor Bang, express that view, and my agreement with those who say it is not safe to rely upon such exposure for the destruction of the

Digitized by Google

tubercle bacillus. For several years I made a number of experiments, at first with the result that it was easy to kill the tubercle bacillus by the exposure of milk or cream to a temperature of 85° C. That is to say, in the experiments I obtained results similar to those of other observers and Professor Bang, therefore I felt justified in supporting the same view as he holds. But of late some doubt arose in my mind as to the safety of the method, and especially as to the safety of sterilising cream at this temperature. I therefore made experiments with the usual care. I mixed cream with tubercle bacilli of human and bovine origin, incorporating, for instance, in one set of experiments tubercular sputum, taking care that every portion of cream was infected and contained tubercle bacilli. I exposed it to various temperatures, and on inoculating guinea-pigs with it I found evidence in 60 per cent. of them of infectivity—not immediately, but after two, three, or four months. If the animals inoculated were kept long enough, it was found that exposure to temperatures of 74°, 76°, 82°, and 85° C. was not in every case sufficient, and I found, to my astonishment, that exposure to a temperature of 85° C. for fifteen minutes delayed the development of tuberculosis in the animal only by one month; that is to say, after two months I obtained in the guinea-pigs evidences of infection equal to those which were obtained in the control animal at the end of one month. Exposure for three hours to a temperature of 65° C. brought about a slight retardation. So from these experiments I came to the conclusion that in the present state of affairs nothing but the boiling of milk is reliable for purposes of sterilisation.

Dr. A. MARMOREK, *Paris*: Je ne voudrais pas laisser passer sans réponse l'explication de la mortalité par tuberculose intestinale, que M. Ruata vient de nous donner en nous montrant cette statistique si intéressante. Sa conception ne me paraît pas justifiée par ces chiffres. Surtout les chiffres élevés de méningite me semblent des plus suggestifs et dirigés contre l'opinion de M. Ruata. On sait que cette forme de la tuberculose n'est pas primaire, qu'elle n'est que la forme finale d'une infection plus ancienne dont les lésions se manifestent dans les ganglions péribronchiques, péritrachéiques ou médiastinaux. De sorte que nous nous voyons obligés de ranger toutes les méningites tuberculeuses parmi l'une ou l'autre forme de l'infection bacillaire, c'est-à-dire, celle par l'air ou par le lait. En dehors de toutes les autres données cliniques ou anatomiques, les chiffres que M. Ruata a mis sous nos yeux apportent beaucoup de vraisemblance à l'hypothèse de l'infection de ces ganglions par le lait infecté; car le nombre très élevé de méningites dans la première année de l'enfant—tandis que cette lésion diminue sensiblement dans la seconde année—nous entraîne à considérer la plupart de ces méningites du premier âge comme causée par une infection due à l'ingestion de lait, une infection qui peut avoir sa porte d'entrée aussi bien dans des glandes des parties supérieures (autour de la trachée et de l'œsophage) que dans celles du canal intestinal.

MR. KINGWELL (*London*), speaking as a butcher, urged the necessity of

Digitized by Google

meat inspection by experienced men, who ought to be bacteriologists. This was the more important in view of the large meat importation into this country.

Professor RUATA, *Perugia*: I would like to repeat that during the first few months of life the death-rate from tabes mesenterica is as high as that for the first twelve months, and, as we know, the numbers having tubercular diseases are small in the first twelve months. Further, tabes mesenterica is never produced in adult life primarily—at least I never saw it. The statistics available are quite in accordance with what we see at post-mortem examinations.

Dr. RAVENEL (in reply) said: Mr. Chairman and Gentlemen,—M. Marmorek has considered and commented on the statistics brought forward by Dr. Ruata, and in doing so has said some of the things I should have said. In further confirmation of what he has brought forward I may mention some of the German statistics for the district around Tubingen, where the people drink boiled milk or goats' milk. If you study the statistics of tabes mesenterica in children you find that they vary in different communities only to a very limited extent. I have been able to trace the connection between the number of cases of tabes mesenterica and the proportion of tuberculous cows in that same community. I do not claim to have been able to do this thoroughly, but in a number of places there seems to be a distinct connection between the occurrence of tabes mesenterica and the existence of tuberculosis in these dairy herds.

THE PRESIDENT: There is one matter upon which I should like the opinion of this Section. We are all aware that there has been a great deal of discussion at this Congress on the question of meat and milk, and the transmissibility of bovine tuberculosis to the human subject. Of course we must accept as something to be very carefully considered anything coming from Professor Koch; but in connection with that, as in connection with the boiling of milk, of which we have heard this morning, we have the practical question remaining as to what is to be done in the meantime, until the question is absolutely settled. We have had a great deal of important evidence brought forward on the opposite side, but we cannot expect that the matter can yet be looked upon as decided. There are many people outside waiting to know what they should do in the meantime, and I should like something of this sort to go forth from this Congress:—

"This Section considers that, in the light of the work which has been presented at its sittings, Medical Officers of Health should continue to use all the powers at their disposal, and relax no effort to prevent the possible spread of tuberculosis by meat and milk."

That commits us to nothing, except that the practical work for the present should be continued, and that no change should be made for the present. It is a matter that will have to be considered. Even Professor Koch considers it should be further investigated. He is satisfied that the results can only be

Digitized by Google

of one kind. Of course we cannot all agree on a point of this kind; it must be considered. But we must, for the present, give some guidance as to what we consider should be done by those on whom the heavy weight falls of acting in this matter. If it is the sense of this meeting, I should like a resolution of this kind to go out as the result of our sittings.

(The resolution as submitted was unanimously agreed to.)

I will now call upon Mr. Foulerton to read his paper.

(Professor HAMILTON, *Aberdeen*, took the chair at this stage.)

Mr. A. G. R. FOULERTON, *London*, read the following paper on "The Influence of Secondary Infections in Chronic Pulmonary Phthisis":—

The influence which is exerted by secondary infections on the course and outcome of certain forms of tuberculosis in man is a matter of the greatest importance in the practice of medicine, and one which began to attract attention so soon as the identification of the specific parasite of the disease enabled its pathology to be studied with exactness. But the difficulty attending on the attempt to arrive at a true solution of this question is no less considerable than its importance; and it was with extreme diffidence that, at the request of the Secretary of this Section, I undertook the responsibility of opening a discussion on mixed infections in tuberculosis.

Having once undertaken the responsibility, however, I felt that by dealing with the one aspect of the question which seemed to me to be of the greatest importance practically, I could better supply material for debate than by merely recapitulating generally the opposing views on this matter which have been put forward by different pathologists—views which are well known to all of you, and which will doubtless be revived and discussed by succeeding speakers.

My own remarks, then, will be limited almost entirely to the consideration of the part played by secondary infections in that form of tuberculosis in which the complication is most markedly manifest, and in which the results of secondary infection are, as it would seem, most disastrous, that is to say, in chronic pulmonary tuberculosis as it occurs in adults.

Chronic Pulmonary Tuberculosis and Chronic Pulmonary Phthisis.

Whilst there can be no question as to the cardinal part which *bacillus tuberculosis* plays as the primary, or original, factor in the condition known as chronic pulmonary phthisis, the influence of the specific parasite in the production of the symptoms characteristic of this condition, and its importance as an ultimate factor in the excessive mortality caused by the disease, are not by any means so well established.

Frequently, indeed almost invariably, the terms chronic pulmonary tuberculosis and chronic pulmonary phthisis are used in clinical medicine as if synonymous; whereas pathologically the two conditions differ widely, although agreeing in the fact that *bacillus tuberculosis* is present in the lesions of each. In uncomplicated chronic pulmonary tuberculosis the single organism present,

Digitized by Google

the specific bacillus, is one which, under the given conditions, does not manifest more than a quite low degree of virulence in the majority of cases, whether as regards its direct action on the tissues actually infected, or as regards its action on the system as a whole by the production of toxins. In chronic pulmonary phthisis, on the other hand, to the action of this parasite is added the more acute influence of pyogenic organisms, whose potentiality for evil—again under the given conditions—is incomparably greater, whether by direct action on the tissues infected or by the production of toxins.

That success in treatment is founded on the correct pathology of a disease is the most obvious of truisms; and since a further reduction of the still excessive mortality from pulmonary phthisis is one of the two great objects to which the efforts of public medicine of the present day are most earnestly directed, the importance of a true appreciation of the influence of the several factors which cause this mortality can scarcely be overstated. In dealing with chronic pulmonary phthisis, are curative measures to be directed exclusively against the specific parasite of tuberculosis, or are we to regard the secondarily infecting organisms as factors of equal, or even greater, importance in the causation of the mortality?

And in seeking an answer to this question, the first thing that one has to consider is, what is the nature of chronic pulmonary tuberculosis before the occurrence of secondary infection has converted the condition into one of chronic pulmonary phthisis?

Uncomplicated Chronic Pulmonary Tuberculosis.

By uncomplicated chronic pulmonary tuberculosis, I understand, is meant the condition in the lungs brought about, usually in adults, by the presence of *bacillus tuberculosis* alone, in which a limited number of granulomata in various stages of development and degeneration are present in the tissues infected, and in which infection has in a large majority of cases occurred by way of the air passages.

The fact that such a condition usually results from direct air-infection is rendered probable by what one observes in the experimental infection of animals.

In lower animals the conditions which correspond with general miliary tuberculosis and with chronic pulmonary tuberculosis can be produced at will, according to the method of inoculation practised. If one causes a general blood infection by the injection of *bacillus tuberculosis* directly into a vein, the result is manifest in the development of a large number of grey miliary tubercles in the lungs—the common seat of arrest and lodgment of bacteria circulating in the blood—in the spleen, and elsewhere. The animal usually dies from the effects of the infection before sufficient time has elapsed for the granulomata to undergo the complete degenerative changes found in cases of tuberculosis in which the degree of primary infection has been less intense.

If, on the other hand, the animal is made to inhale infected dust, one gets a chronic, and at first strictly localised, tuberculosis, such as is the first stage

Digitized by Google

towards chronic pulmonary phthisis. Under these conditions, unless the initial dose of bacteria inhaled has been excessive, life is sufficiently prolonged for the resulting granulomata to undergo the successive degenerative phases seen in the lesions of chronic pulmonary tuberculosis in man.

The first thing to be noted about uncomplicated chronic pulmonary tuberculosis in man is that the condition is comparatively rarely met with by the physician in his practice, and that the opportunity for the study of its morbid anatomy is found mainly in patients who have died from some other disease. It is rarely recognised by the physician because of the absence of any definite symptoms produced by it in the majority of instances, such cases as are diagnosed being usually those in which a certain amount of cough has attracted attention to the chest, and the sputum has in consequence been examined. In such cases physical signs and definite symptoms of disease are most frequently absent; and the discovery of the infection by the finding of bacilli in the sputum is usually almost a matter of accident.

On the other hand, such a condition of uncomplicated chronic pulmonary tuberculosis, as being the initial stage of chronic pulmonary phthisis, must, we know, be an extremely common one; and the frequency of its occurrence is emphasised when we consider, not only the number of cases in which secondary infections have determined the onset of definite pulmonary phthisis, but also the large number of cases of pulmonary tuberculosis which there is reason to believe never arrive at that stage, and are not recognised at any time during life. The frequency of these latter cases can only be estimated from the number of instances in which lesions, usually of limited extent, indicative of cured or arrested tubercular infection, are found in the lungs of adults dying from other causes.

Ample evidence of this has been brought forward by many pathologists, and I need now only quote the following facts.

The condition of the lungs with regard to evidence of past disease has been recorded with particular care in the case of all patients dying with malignant disease in the cancer wards of the Middlesex Hospital, during the past eighteen months. In all one hundred and twenty-eight such cases have been examined during the period, and the following results have been tabulated:—

Age.	Lungs free from any lesions suggestive of recent or cured tuberculosis.	Lungs with lesions suggestive of recent or cured tuberculosis.
1–20	3	—
21–30	2	—
31–40	6	3
41 and over . . .	80	34

The lesions found may be roughly classified as follows:—

Recent localised tuberculosis, 3 cases.
Caseous or calcareous nodules in lungs, 18 cases.
Cicatrisation of lung substance, 16 cases.

Digitized by Google

In all these cases the extent of the lesions was limited; and in not one case was any actual history of past phthisis obtained during life. Of the cases in which lung lesions of one sort or another were found, there was absolutely no history of any chest trouble at all in thirty-four; in the case of the remaining three the following histories were given:—One patient had suffered from "inflammation of the lungs," and another from "chronic bronchitis," both some years previously; whilst the third, a woman aged 34, stated that she had suffered from "spitting of blood" seventeen years previously. Seven of the ninety-one patients who, after death, showed no lesions in the lungs had also given a history of either "bronchitis" or "inflammation of the lungs" at some time previous to admission.

Excluding the three cases of recent tubercular infection, the result of the investigation shows that of one hundred and twenty patients of both sexes who had survived beyond the age of thirty, thirty-four showed definite evidence of past destruction of lung substance, with subsequent arrest of the disease which caused it. And knowing from post-mortem examinations the relative frequency of occurrence of diseases which cause such destruction of lung substance, it cannot be questioned but that a large proportion of these thirty-four patients had at one time or another suffered from pulmonary tuberculosis.

Dr. Lazarus-Barlow has been kind enough to give me the results of the last one hundred and sixty-two consecutive post-mortem examinations which he has carried out at the Westminster Hospital on adults over the age of thirty dying from diseases other than tuberculosis. Of one hundred males, eighteen showed evidence of arrested tuberculosis; and of sixty-two females, fourteen showed similar evidence. This gives a proportion of thirty-two cases of arrested tuberculosis in one hundred and sixty-two of both sexes. We find, then, that in individuals dying from causes other than tuberculosis a considerable percentage show evidence of having at one time or another suffered from a localised pulmonary tuberculosis, which has not been sufficiently severe to attract notice during life by definite symptoms. If we compare the ratio of deaths from chronic pulmonary phthisis to deaths from all other causes, other than the various forms of tuberculosis, in adults over the age of thirty, we can gain some idea as to the frequency with which the arrest and cure of chronic pulmonary tuberculosis occurs without a development into pulmonary phthisis.

We next have to consider the nature of the bacteria which, by infecting the lesions of chronic pulmonary tuberculosis, convert that condition into one of chronic pulmonary phthisis.

The Secondarily Infecting Bacteria of Chronic Pulmonary Phthisis.

Our knowledge of the flora of pulmonary tubercular cavities has been arrived at by the examination of (1) the sputum discharged during life, and (2) of the walls and contents of the cavities after death.

Digitized by Google

Examination of the sputum has usually been carried out after the plan suggested by Kitasato—the mouth is well rinsed out, the sputum caught in a sterilised vessel, and washed repeatedly with sterilised distilled water. Culture media are then inoculated with the sputum and incubated.

Using this method, the error arising from contamination by bacteria from the mouth and pharynx is reduced as much as possible, and on comparing the results of the cultures with stained specimens of the sputum, a reasonably accurate estimate of the relative proportion in which any bacteria are present in the pulmonary lesions during the progress of the disease may be obtained. Cultivation of the bacteria found in the contents of the cavities after death, and microscopic examination of the cavity walls, appear to me as a method of examination of rather less value than the preceding; the results obtained are, so far as the particular object in view is concerned, very liable to be vitiated by the occurrence of bacteria which have infected the cavities only during the last few hours before death, or afterwards, and which consequently have had no influence on the progression of the disease during life. A full catalogue of the literature embodying the work of those who have dealt specially with the species of bacteria which infect such cavities has lately been published in Sata's monograph on "Mixed Infections in Pulmonary Phthisis,"* and there is no need for me now to do more than mention the different species commonly found.

Practically all who have worked at the subject are agreed that various species of cocci are the predominating organisms in the secondary infection of tubercular cavities, *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *Staphylococcus pyogenes albus*, *Diplococcus pneumoniae*, and *Micrococcus tetragenus* being found more especially. Amongst other bacteria of less common occurrence under these conditions are *Bacillus coli communis*, *B. pneumoniae* of Friedländer, *B. pyocyaneus*, species of *Proteus*, and *Saccharomyces albicans*. Sata also describes *B. pseudodiphtheriticus* and *B. pseudodiphtheriticus pulmonalis* as occurring with some frequency.

Artault,† again, has compiled a very full list of the bacteria which are found in cavities after death, but many of the organisms mentioned by him, and by others who have carried out this method of examination after death, must have been present as merely casual contaminations, and cannot be considered seriously as having had any definite influence on the course of the disease.

It is to the pyogenic cocci, and above the rest to *Streptococcus pyogenes*, that we have therefore to look when considering the effects of secondary infections as exercised through the lengthened course of a case of chronic pulmonary phthisis.

---

* Sata: "Ueber die Bedeutung der Mischinfection bei der Lungenschwindsucht." Jena, 1899.

† Artault: "Flore et Faure des Cavernes Pulmonaires." *Archives de Parasitologie.* Tome 1, p. 217. 1898.

Digitized by Google

The Pathogenic Action of the Secondarily Infecting Bacteria.

It may next be asked in what manner it is that these cocci exert an injurious influence—a point which must be dealt with in some detail. And the action of such organisms may be considered, first, as to their action locally on the tissues actually infected, and secondly, as to their effects on the system generally.

The local effects are manifest by an active destruction of the tissues actually infected, by the occurrence of perinodular pneumonia, and by what may be described in general terms as a lowering of the powers of resistance—a lowering of the vitality of tissues in the neighbourhood of those actually infected; the latter effect being partly due to the direct action of toxins produced by the bacteria, and partly to vascular disturbance in the proximity of the lesions.

Their action on the system at large may be expressed either by the results following on the absorption of toxins (toxæmia), or by those of an actual infection of the blood generally by the bacteria themselves.

From these ways in which the secondarily infecting bacteria might act injuriously, we may, I think, exclude a general blood infection as being a factor of any importance in the pathology of chronic pulmonary phthisis, save only as a terminal infection.

Evidence on this point is afforded by examination of the blood during life, and of the blood and organs after death.

Petruschky * examined the bodies of fourteen patients who had died with pulmonary phthisis, and in eight cases isolated streptococci from the blood and internal organs. These observations are of value only as establishing the relative frequency in phthisis of terminal infection by the organism which is the predominant secondarily infecting parasite during life, and beyond that have no direct bearing on the pathology of the progressing disease.

Jakowski † examined blood, drawn from the finger, from patients in the hectic stage; in eight cases out of nine thus examined, Jakowski obtained cultures, either pure or mixed, of *Staphylococcus pyogenes aureus*, *Staphylococcus pyogenes albus*, or *Streptococcus pyogenes*. The method of examination used has, however, now become obsolete because of the difficulty of avoiding contamination, and in the light of recent work Jakowski's results must be looked upon with suspicion. Thus Straus,‡ on making cultures from the blood of phthisical patients, "en pleine fièvre hectique," the blood being obtained with a syringe directly from a vein, found it absolutely sterile in every case out of thirteen examined.

---

* Petruschky: "Tuberculose und Septicämie." *Deutsche Med. Wochensch.* 1893. S. 317.

† Jakowski: "Beitrag zur Frage über die sogenannten Mischinfektionen der Phthisiker. Untersuchungen des Blutes der Phthisiker in der hektischen Periode." *Centralblatt für Bakteriologie, Band xiii., S.* 762. 1893.

‡ Straus: "Tuberculose et infections secondaires." *La Semaine Médicale.* 1894. p. 253.

Digitized by Google

Mangin-Bocquet * similarly examined the blood from seven cases of advanced phthisis, one cubic centimetre of blood being withdrawn from a vein and transferred to culture media. In five out of the seven cases the culture tubes remained sterile ; in the other two cases a small proportion of the tubes inoculated showed a growth of a white coccus.

The balance of evidence afforded by these and other observations allow us, then, to exclude general blood infection as a factor of any consequence in the present discussion. And the effect of secondarily infecting organisms has to be considered only with regard to their local action and with regard to the production of a toxæmia.

Method of Secondary Infection.

The method by which secondary infection occurs may just be mentioned. In normal tranquil respiration, carried on through the nostrils, all bacteria in the air inspired are arrested in the anterior part of the nasal passages, and so the air which passes through the glottis into the lungs is under ordinary circumstances free from organisms.

This arrangement is necessarily disturbed by the act of coughing, when a sudden and deep inspiration through the mouth occurs preparatory to violent expiratory efforts. And under these circumstances potentially pathogenic bacteria will pass with the rush of air through the glottis, and into the lungs, the working of the normal mechanism for their arrest being in abeyance.

Assuming that the lining cells of the lower portion of the respiratory tract are in a healthy condition, and that the dose of inspired bacteria is not excessive, the natural germicidal power of the healthy mucous secretion will probably be sufficient to destroy the activity of any invading bacteria before mischief is done. If, however, portions of the mucous lining have their vitality impaired by the irritation caused by neighbouring tubercular nodules, or if granulomata have, in breaking down, exposed small cavities containing caseous material, any invading bacteria find conditions favourable for their growth, and are able to effect a lodgment.

It is of some interest to note that the bacteria which are under normal conditions arrested in the fore part of the nasal passages are amongst those which are most frequently found infecting pulmonary cavities. Thus Von Besser,† on examining the nasal secretion of fifty-seven individuals, found *Diplococcus pneumoniae* present fourteen times, *Staphylococcus pyogenes aureus* fourteen times, *Streptococcus pyogenes* seven times, and *B. pneumoniae* (Friedländer) twice.

Apart from the infection of the lungs by inspired air, one must just mention secondary infection passing down the trachea by direct continuity of surface, in cases where there is a laryngitis, as a possible mode of secondary infection of tuberculous lesions in the lungs.

---

* Mangin Bocquet: "De la Fièvre dans la Tuberculose et principalement de la Fièvre hectique." *Thèse*, Paris. 1896.

† Von Besser: "Ueber die Bakterien der normalen Lustwege." *Centralblatt für Bakteriologie, Band vii, S.* 151. 1890.

Digitized by Google

Comparison of the Pathogenic Action of B. tuberculosis and the secondarily infecting bacteria in the production of the characteristic phenomena of Chronic Pulmonary Phthisis.

Uncomplicated chronic pulmonary tuberculosis being a condition which, in a large majority of the cases in which it occurs, does not of itself give rise to serious symptoms, and which, in a probably considerable number of the cases in adults, appears to become spontaneously arrested, we have next to consider to what extent the serious and fatal features of chronic pulmonary phthisis are due to the further growth of *B. tuberculosis* and to the secondarily infecting bacteria respectively.

Taking the primary pathological features of fully developed chronic pulmonary phthisis during life as including (1) evidence of active destruction of lung tissue, and (2) the production of what is termed "hectic fever," with its accompanying high evening temperature, morbid sweating, and wasting of the tissues generally, we must at the start allow that infection of a cavity by, for instance, *Streptococcus pyogenes* will afford a satisfactory explanation of all the phenomena. On the other hand, we find good reason for believing that *B. tuberculosis* by itself would not—at any rate, under the conditions which prevail in what may be termed a "chronic infection," such as we are now considering—be capable of producing these effects in anything like the extent to which they occur. As to active destruction of tissues, this is never a direct effect of a pure infection by *B. tuberculosis*. A slow destruction of tissue does, of course, occur, but only because of the breaking down of granulomata, the very presence of which, as they occur in tuberculosis, is evidence of a low grade of virulence in the parasite producing them. The organism comes to rest at some point in the tissues, and its presence there is just sufficient to determine an aggregation of round cells, from which the granuloma is developed. A quite similar result may be produced by a purely mechanical irritation, and up to the time when the granuloma is fully developed, there is, so far, no evidence whatever of any active destruction of tissues. The subsequent degenerative changes which the tuberculous granuloma may undergo are to be attributed quite as much to a cutting off of the blood supply of the cells concerned as to any injurious action on the cells by the bacteria contained in the lesion; and with these changes there is no active destruction of the living tissue neighbouring on the caseating or breaking-down granuloma, but merely a certain amount of round-cell infiltration, which, in the neighbourhood of the simple tuberculous granuloma, is always of slight extent.

With regard to the formation of toxins, which by a toxæmia might produce general or constitutional symptoms, all that one can say is that in all forms of chronic pure tuberculosis in parts other than the lungs, the symptoms which can be attributed to such a toxæmia are either of the slightest or conspicuously absent. When one does get symptoms due to an acute infection by *B. tuberculosis* they are of a quite different character, not merely in degree, but in type, from those of chronic pulmonary phthisis. The comparative inertness of chronic tuberculous disease, before an entirely new feature has

Digitized by Google

been added by secondary infection, cannot be better illustrated than by what occurs in cattle. An ox may have the most extensive tuberculous infection of the pleural or peritoneal sacs, and yet will frequently be in absolutely prime condition; there is no suggestion of disease until the tuberculin test is applied, or the animal is slaughtered. But given a breaking down of tubercles in the lung substance, with secondary infection of the cavities, or an ulceration of tuberculous lesions in the intestine, and one finds the same high temperature, the general wasting, and the same active destruction of the infected tissue which characterise the average case of chronic pulmonary phthisis in man. In cattle the effects of tuberculosis depend not on the extent of the specific lesions—so long at least as these are localised—but rather upon the occurrence of secondary infection. When one considers more especially those forms of chronic tubercular infection which come more commonly under the care of the surgeon, one finds the same thing; so long as secondary infection by pyogenic bacteria is absent, constitutional symptoms indicative of any serious toxæmia are wanting. When a secondary infection occurs, either spontaneously or as the result of operative interference, one gets the same train of general symptoms as those which mark the transition of uncomplicated pulmonary tuberculosis into chronic pulmonary phthisis.

Believing, then, that secondary infection of the lungs is the chief, or at any rate the immediate, cause of the excessive mortality from pulmonary phthisis, it is permissible to ask whether the pathology of these infections can throw any light upon their treatment.

The Treatment of Secondary Infections in Pulmonary Phthisis.

Stated generally, I believe that the teaching of pathology on the matter is that the treatment of secondary infections should be the chief object aimed at in the first place.

Believing strongly as I do that ultimate success in the specific treatment of tuberculosis will be attained with some modification of tuberculin, I also feel that the comparative failure of the method in the past, and the consequent discredit which has fallen on it, have been almost entirely due to the ignoring of the effects of the common secondary infection of the lung lesions by pyogenic cocci. Knowing as one does the intense reaction in the neighbourhood of tubercular lesions which may follow the use of tuberculin, it is difficult to understand how its use in a patient whose lesions are secondarily infected and lung tissue damaged by actively virulent pyogenic bacteria could fail to be followed by disastrous consequences. Before any attempt at the use of such a method should be made, the extent of any secondary infection should be carefully taken into consideration. And as a very large proportion of the cases which come under treatment will have already suffered this secondary infection, the question must arise as to whether any practical steps can be taken to reduce the intensity of such an infection by pyogenic cocci. And I think that one can fairly claim that in cases of average severity efficient steps might be taken in this direction. By keeping a patient who has a cavity

Digitized by Google

infected by pyogenic cocci in an atmosphere of practically germ-free air, such as may be found at moderate altitudes, and by enforcing the nearest approach to complete physical rest possible, one can not merely protect him against the risk of reinfection by pus organisms, but one also places him under conditions in which the work which has to be done by a damaged lung is reduced to a minimum. With the patient thus protected as far as possible against any reinfection of the cavity, one may confidently hope that the intensity of the present pyogenic infection will be reduced. It is a matter of common experience that when pyogenic bacteria are confined in a limited space, and are kept from reinforcement from outside, their virulence becomes attentuated, and that after a time they tend to die out. Thus the streptococcus from an old empyema, old pyosalpinx, or chronic abscess of any sort will often be found to have lost much, or all, of the virulence which it originally must have possessed. Charrin and Guillemonat* have recently shown that the same attenuation of virulence in intestinal bacteria takes place when an animal, without any special preparation, is kept in an atmosphere of sterile air and fed on sterilised food. In the absence of reinforcement from without, the number of intestinal bacteria is markedly diminished, and the vitality of such as survive is distinctly lowered. If, therefore, a patient is kept under the conditions which I have spoken of, one may expect that improvement, so far as the pyogenic infection of cavities is concerned, will follow. And it is probable that this is what happens in most of the cases in which apparent arrest of phthisis follows treatment in well-ventilated hospitals and sanatoria. Everyone knows, however, the common after-history of such cases. A working-man as the result of such treatment puts on weight, night sweats disappear, the physical signs become insignificant, and as I said the tuberculosis is looked upon as being arrested, or even cured. What has probably really happened is that, with the disappearance of secondary infecting bacteria, the pulmonary phthisis has reverted to the former condition of uncomplicated pulmonary tuberculosis. If the patient returns to his old surroundings, a reinfection with pyogenic cocci from the foul air in which he lives occurs, and the case slowly drifts back into one of chronic phthisis again. Or if the conditions are more favourable, the proper tuberculous lesions may in time undergo a natural cure in the absence of any reinfection.

The constancy with which a recrudescence of the disease happens in patients with arrested phthisis discharged from sanatoria and hospitals to their old surroundings cannot be attributed to reinfection with *B. tuberculosis*, and no explanation other than a reinfection of tuberculous lesions with pyogenic organisms will suffice.

I have laid some stress upon this matter, because it is just in this stage of arrested phthisis that specific treatment of the now more or less pure tuberculosis with tuberculin will have the best chance of success. The lungs being free from pyogenic organisms, the result of the specific reaction might be

* Charrin and Guillemonat: "Comptes Rendus de l'Académie des Sciences." Tome cxxxii., p. 1074. 1901.

Digitized by Google

expected to be far less severely felt, and a real cure of the tuberculosis may be hoped for.

In addition to the treatment by rest and pure air, it is likely enough that a systematic treatment with anti-streptococcic serum in cases in which *Streptococcus pyogenes* is the predominating organism—and those cases form a large proportion of the whole—would help materially in shortening the time which must elapse before any specific treatment for the tuberculosis can be safely attempted.

Other problems of interest in connection with the subject of mixed infection in pulmonary tuberculosis, which I have not had time to touch on, are (1) the effect of secondarily infecting organisms on the progress of the tubercular infection itself, a question on which diverging views have been expressed, some holding that the growth of the bacillus is thereby checked, and others that its dissemination is favoured; and (2) the exact pathology of those cases in which phthisis rapidly supervenes after some infective diseases, such as typhoid fever, influenza, or variola. Are such cases the result of secondary infection of a lung already the seat of latent tuberculosis, or are they the result of a secondary infection by *B. tuberculosis* of a lung already damaged by bronchitis or pneumonia in the preceding specific disease?

MIXED INFECTIONS IN PULMONARY TUBERCULOSIS.

By Professor W. Ophüls, *San Francisco.*

In this paper I shall confine myself to a study of the effects of mixed infection on pulmonary tuberculosis, because as far as mixed infection in other tubercular lesions is concerned I have very little personal experience, and the literature on this subject seems to be extremely meagre.

The possible importance of mixed infection in pulmonary tuberculosis suggested itself very early, which is only natural when we consider how much the large wounds that are produced in the lungs in this disease are exposed to secondary infection from the outside. Time will not permit me to enter here into a full discussion of the historical development of this interesting question, and I feel at liberty to review the literature on the subject very shortly, since in the monograph of Ortner, and the more recent publication of Sata, we find a full discussion of this part of the subject.

In a general way we can say that the subject has been approached in two different ways, the examinations of one class of authors being largely bacteriological—bacteriological examinations of the washed sputum of living patients, bacteriological examinations of the contents of cavities and the material obtained at autopsy from suspected areas of mixed infection. It is true that the majority of these authors supplemented their purely bacterio-

Digitized by Google

logical research by histological investigations, but in many cases the latter certainly have not received sufficient attention. Bacteriological methods pure and simple, under such complex conditions, are very apt to mislead. The bacteriological examination of the sputum at best tells us simply of the bacteriological flora in the pulmonary cavities, and even the direct bacteriological examination of the lungs at the post-mortem is very deceptive, on account of the difficulty of recognising whether the bacteria which grow in our cultures really are directly responsible for the lesions at the points from which the culture was taken. In fact, as Ortner already pointed out, the histological investigation is the much more important one, because it not only allows us to recognise the presence of other bacteria besides the tubercle bacilli, but also shows us their direct relation to the diseased process. It is only by the combination of bacteriological and histological methods that we can hope to arrive at the truth.

As far as the present status of our knowledge on the importance of mixed infection in pulmonary tuberculosis is concerned, the general consensus of opinion seems to be that its effect upon the course of the disease is certainly a very profound one, but just how far it is responsible for the acute exacerbations of the disease which we observe so frequently in our patients is much more doubtful. Some authors (Ortner, Schabad, many of the French authors, and even Sata) go so far as to ascribe all, or nearly all, acute processes in pulmonary phthisis to mixed infection, whereas others (Fränkel and Trojé, for instance, who certainly have proved the occurrence of acute pneumonic processes of purely tubercular nature) do not altogether deny the occurrence of mixed and secondary infections, but are inclined to believe them of little importance in the development of the disease. We can fully appreciate the widely divergent views which, up to the present time, are held on this subject, when we on the one hand read in an editorial of the *British Medical Journal* of July 28, 1900, this statement, under the caption "The Rôle of Streptococci in Phthisis": "It is a remarkable fact that . . . . the bulk of the distressing and dangerous features of phthisis pulmonalis are not due to the tubercle bacilli, but to the streptococci or other pyogenic organisms. The fever, the excavation and the expectoration, the waxy degeneration of the viscera, and in great part the diarrhœa, are all to a greater or less extent due to streptococcus infection. It may be said, indeed, in a general way, that the risk of phthisis *quoad vitam* is not a matter of tubercle bacilli . . . ." On the other hand, Schroeder and Mennes, who have written one of the latest monographs on the subject, after finding pyogenic cocci in the washed sputum of all consumptive patients, and after pointing out their small virulence or complete lack of virulence when tested by the animal experiment, come to the conclusion that the pyogenic cocci do not have any, or very little, influence on the tubercular process, and have to be regarded more as saprophytic organisms commonly encountered in the lungs in this particular disease. The truth, of course, as usual, lies in the middle, and it is not difficult to show that some of the acute exacerbations, which usually assume the form of pneumonic processes, are produced by the tubercle bacillus alone, whereas others are the result of

Digitized by Google

mixed infection, a solution of the difficulty which has been suggested by Orth in his contribution on caseous pneumonia as early as 1891.

What remains to be established is, in the first place, the relative frequency of the one or the other form, and, secondly, the direct influence either of them has on the progress and eventual fatal termination of the disease.

These two questions can be answered only in one way. In the first place, a study of consecutive, not *picked*, cases is necessary, and it is also necessary to exclude from this study all forms that do not belong directly within the range of pulmonary phthisis—that is, for instance, acute disseminated miliary tuberculosis originating outside the lungs with pneumonic complications. In the second place, these cases must be studied in their entirety. It is not sufficient to confine one's research to the lungs, or even less to one particularly interesting spot in the lungs, but all important organs have to be examined carefully with the naked eye, with the microscope, and bacteriologically, otherwise it is impossible to estimate the bearings of the pulmonary process on the general health of the patient. The lungs naturally will receive special attention, and here an attempt should be made, on the basis of microscopic and macroscopic finding, to distinguish between old and recent changes, in order to reconstruct, as far as possible, the course of the disease from the findings on the post-mortem table. I am fully aware of the difficulty of the task, and I am ready to acknowledge that my first efforts in that line were little satisfactory, even to myself; but in course of time I imagine to have seen clearer, although, of course, my views necessarily must be more or less individualistic and need correction from other sources.

According to this plan, I have endeavoured to study as well as possible a series of thirty-nine consecutive cases* of phthisis pulmonalis, and have added to them a fortieth case, that came to autopsy only recently, on account of its special importance. It is unnecessary to emphasise that, in the study of such a comparatively large number of cases, one has to use one's judgment as to what to examine and what not, otherwise one life would not be long enough to make a complete examination. As far as the pulmonary lesions are concerned, it is better, I believe, to examine carefully selected spots thoroughly than attempt an examination of all diseased areas. For instance, I did not examine every time the old fibroid tubercles that were found, because repeated examination had convinced me that they never contain any organisms besides tubercle bacilli.

At the end of the paper I give a short synopsis of the forty cases examined, with some abstracts of the clinical histories as far as they could be obtained. For these latter I am very much indebted to Dr. J. O. Hirschfelder, under whose care most of the patients were, and to my assistant, Dr. W. O'Connor, who collected and abstracted them for me.

In all cases a complete post-mortem record was taken, only the central nervous system was not examined in all cases. Then routine histological

* These cases have been made use of already by me in my paper on "Pneumonic Complications in Pulmonary Phthisis" in the 'American Journal of Medical Sciences,' July, 1900.

Digitized by Google

examinations were made shortly after the post-mortem of all important organs. Routine bacteriological examinations of interesting spots in the lungs, and of the spleen, liver, and kidneys, were also made in all cases, with the exception of cases 7 and 33, in which only lung and spleen, of case 14, in which only lung, spleen, and kidney were examined, and of cases 9 and 15, in which no bacteriological examination was made. The method used for obtaining material for the bacteriological examination was the following:— First the cut surface of the organ to be examined was seared with a hot iron at the selected spot. Into the burnt area, which should be perfectly dry, an incision was made with a hot knife, and then a stout platinum loop punched into the organ a little distance through the incision. The material obtained in this way was carefully spread over the surface of a slanting tube of glycerin agar-agar, and the culture then incubated. I believe that this technique—for which, of course, I do not claim any originality—has many advantages over that employed by former investigators on our subject. At least, I have rarely met with contaminations except the unavoidable colon bacilli, have nearly always obtained the development of separate well-characterised colonies, and have found bacteria in lesions which afterwards, on histological examination, proved free from them rarely, when the histological examination did not furnish some explanation (adjoining areas with mixed infection, small bronchi with mixed infection in them). Later on, thin sections of interesting parts of the lungs were made, and stained for tubercle bacilli and other bacteria (alkaline polychrome methylene blue, dilute carbol-fuchsin, Gram-Weigert).

The lesions that were found in the diseased lungs may be shortly described as follows:—

1. Old cavities, present in all cases except No. 40. Their characteristics are too well known to need further description. I should like to state that all cavities that I comprise under this heading have a distinct wall of granulation and fibrous tissue (third stage of Sata).

The contents of twenty-six old cavities were examined in thirteen cases. A pure culture of tubercle bacilli was found in seven cavities; tubercle bacilli and streptococci in one cavity; tubercle bacilli and pseudo-diphtheria bacilli in four cavities; tubercle bacilli and pneumococci in six cavities; tubercle bacilli, pneumococci and pseudo-diphtheria bacilli in four cavities; tubercle bacilli, staphylococci and streptococci in two cavities; tubercle bacilli, pneumococci and staphylococci in one cavity.

2. Tubercular scars. These are irregular, usually quite extensive areas of cicatricial tissue, in which one usually finds numerous tubercles, mostly in a fibroid condition. Such scars, however, may contain a considerable number of caseated tubercles and quite recently formed small cavities.

3. In the surroundings of old cavities and old tubercular scars, one very frequently finds a condition which Rindfleisch has very appropriately called "inveteriertes Oedem." These areas are more or less indurated, do not contain any air, present a smooth grey cut surface, in which, on closer inspection, one sees many bright yellow dots. Microscopically one finds the septa more or less thickened and indurated, the air spaces filled with desquamated epithelial

Digitized by Google

cells full of fat granules and oedematous fluid. There may be considerable fibrin in these areas. I have never found bacteria in them and have neglected them somewhat. They are much more frequent than would appear from my protocols.

4. Old tubercles, either disseminated or in conglomerate form. In conglomerate tubercles we frequently find a pigmented scar in the centre showing the tendency for these lesions to heal. In spite of this the progress in the periphery may be quite rapid, as is shown in case 34, in which we have a central scar surrounded by tubercles, with extensive caseation and many tubercle bacilli in the caseous areas. Such conglomerate tubercle may also caseate in the centre, break down, and give rise to the formation of new cavities. I have never found any other bacteria but tubercle bacilli in conglomerate tubercles, unless it be in the cavities in them, to which they can easily gain access secondarily.

5. Areas of what I have proposed to call tubercular pneumonia, that is, pneumonic processes in which only tubercle bacilli are found at the post-mortem. I now believe it is best to distinguish an acute, subacute and chronic form. Histologically they correspond to Sata's "unabgegrenzte und abgegrenzte käsige Bronchopneumonie."

Macroscopically the changes begin with a gelatinous infiltration, usually of dark red colour; later the centres of the lobuli become more firmly consolidated, and may present a more or less projecting, more or less granular cut surface, with variations in colour from dark red to greyish red and grey; then follows a central caseation, and in the more chronic forms a more or less marked induration of the tissues around the caseous areas. The caseous areas usually have a marked tendency towards softening, and irregular new cavities are frequently formed quite early in the process.

Histologically we have in the acute form a central caseation involving the infundibulum and nearest air spaces, surrounded by air spaces in which one finds much fibrin, epithelioid cells, lymphocytes, a few desquamated epithelial cells, and a varying number of polymorphonuclear leucocytes. The more acute the process, the more polymorphonuclear leucocytes are usually encountered. In case 40, in which we have a confluent, very acute tuberculous pneumonia involving both entire upper and the entire middle lobe, some air spaces are filled almost exclusively with fibrin and polymorphonuclear leucocytes. They contain enormous numbers of tubercle bacilli, and no other bacteria.

I believe that the epithelioid cells are derived from the pre-existing connective tissue cells of the septa and wander into the air spaces just like migrating leucocytes, because in the early stages of the process they are found entirely separate from the wall in the middle of the fibrin. Later, while they remain in the same position, fine connective fibrilli appear near them, which, according to our present knowledge, proves pretty definitely that they must be derived from connective tissue cells, and are not emigrated large mononuclear leucocytes.

The peripheral air spaces contain more desquamated epithelial cells and lymphocytes, and there is less fibrin and more oedema.

Digitized by Google

In many cases the exudate is distinctly hæmorrhagic, and in the more acute forms the capillaries in the septa that have not as yet undergone caseation are full of blood.

In the more chronic cases the number of epithelioid cells is greater, and the production of connective tissue fibrils more marked. Langhans' giant cells may appear. The septa also begin to show evidence of fibrous thickening, and in the very chronic cases septa and contents may in the surroundings of the caseous material fuse together into a more or less homogeneous mass of tubercular granulation tissue ("abgegrenzte käsige Bronchopneumonie": Sata).

The softening, which leads to the formation of new cavities in these areas, invariably begins in the centre of the caseous areas, and very frequently is preceded by a more or less marked infiltration of the caseous material at the point of softening with polymorphonuclear leucocytes, which nearly always rapidly degenerate and furnish a considerable amount of nuclear *débris*. I have observed this accumulation of polymorphonuclear leucocytes in cases in which only tubercle bacilli were present, and also in those cases in which there appears to be a fresh secondary infection of the caseous material by way of the bronchial tube. At least this seems to me to be the only way in which the presence of pyogenic cocci can be explained in the centre of an area of tuberculous pneumonia, in which, otherwise, one finds tubercle bacilli only. Such secondary infections, however, I did not find very frequently. I have observed them in cases 2, 29, 19, 27, the pyogenic organisms in three cases being pneumococci, in one staphylococci. The pyogenic cocci are certainly not necessary for the production of softening, and I also cannot affirm very positively that when they are present the softening progresses very much more rapidly. Indeed, the tubercle bacilli seem to play a much more important part in the process. In most cases they are present in these spots in very large numbers; I have, however, exceptionally found only a few tubercle bacilli in an area with marked signs of a liquefaction of the caseous material. It also appears that the gathering of leucocytes, which I already alluded to, takes place most markedly when the tubercle bacilli are most numerous, or when pyogenic cocci are present, but I have also seen exceptions from this rule.

In the forty cases which I examined, I have found areas of tubercular pneumonia very frequently; in all, in twenty-five cases. In one case (40) there was a confluent recent very acute tubercular pneumonia which extended over both entire upper and the entire middle lobe, and undoubtedly was the direct cause of death. This case, however, does not belong to my series of consecutive cases. Of these latter, four cases (34, 2, 27, 26) showed acute lesions. In one of them (34) the acute pneumonia changes were found around conglomerated nodules, in the caseous parts of which exceptionally many tubercle bacilli had developed, and after the breaking down of the caseous material had then been directly discharged into the surroundings. In four more cases (1, 13, 22, 35) acute and subacute changes were combined. Subacute lesions were present in three cases (8, 29, 13), subacute and chronic

Digitized by Google

ones in three (33, 36, 32), chronic ones in eight (37, 3, 14, 16, 18, 25, 9, 31), and acute and chronic ones in two (19 and 15).

The affected areas varied considerably in size. The more acute the process the more extensive the affected spots were, in most cases, but this is not by any means invariably so. The areas are either scattered, distinctly lobular, or they may also fuse together and take up large portions of a lobe, or in exceptional cases one or more entire lobe.

I believe that in all cases, except case 34, these areas of tubercular pneumonia were caused by an aspiration of the contents of old cavities. I have not been able to find places—such as they are described by Sata—where the wall of cavities had been partially destroyed, and the infectious material scattered directly into the adjoining healthy tissue through the opening thus produced. I have, however, devoted much less attention than Sata to the study of the walls of cavities in the different stages of their development.

The areas of tubercular pneumonia were usually situated in the lower lobes, or near old cavities, which latter fact can be easily explained by the assumption that sometimes the bronchi leading from them become so much choked with the discharged contents that part of them run into some of the branches, usually those nearest the old cavity.

6. Examples of recent mixed infection I have found in twelve cases. In a majority of the cases (seven in all) the lesions presented the appearances of an inflammatory oedema which could be differentiated from an ordinary oedema due to stagnation only by careful histological and bacteriological examination. Histologically one finds evidence of a beginning emigration of polymorphonuclear leucocytes, and a bacteriological examination of the sections shows in all instances pyogenic cocci in very large number. Tubercle bacilli also were invariably found in the sections; sometimes only a few, but often a considerable number.

It is worthy of note that simple stagnation-oedemata were found only in three cases; once (4) locally in the surroundings of conglomerate tubercles, twice (24, 26) with a more extensive distribution in the posterior parts of the lower lobe. The bacteria found in the inflammatory oedema, together with the tubercle bacilli, were twice (22, 15) pneumococci; once (7) pneumococci and streptococci; once (12) pneumococci and staphylococci; once (17) pneumococci and non-identified bacilli; once (20) streptococci, and in a last case (35) streptococci and staphylococci.

In one case (15) the lesion produced by the acute mixed infection presented the picture of a catarrhal pneumonia. Pneumococci and tubercle bacilli were found in this case.

In three cases the lesions were those of an acute broncho-pneumonia. In two of them (19, 28) pneumococci and tubercle bacilli were present, in one (11) streptococci and tubercle bacilli.

In the last case (13) the broncho-pneumonia showed a hæmorrhagic character, and in it streptococci were associated with the tubercle bacilli.

There was, furthermore, one case (36) in which the lesions produced by the mixed infection were of a more chronic nature, the sections showing a

Digitized by Google

hæmorrhagic broncho-pneumonia with beginning carnification. In the sections which were stained for bacteria only a few tubercle bacilli and no other bacteria could be found; the culture experiment, however, revealed the presence of a limited number of pseudo-diphtheria bacilli.

In many of the cases the tubercle bacilli which were usually found in the infundibula were surrounded by a few epithelioid cells, and in some there was evidence of a just beginning caseous degeneration of the alveolar content in their surroundings.

In six cases the local acute mixed infection was followed by a more general infection. In two cases (31 and 21) pneumococci, besides in the lungs, were also present in the liver; in one (35) they were found in the spleen, in another one (20) streptococci were present in the spleen, in yet another one (12) staphylococci in the liver, and in case 11 the streptococci had infected all serous membranes (pleura, pericardium, peritoneum, meninges), and had also entered the blood and caused a streptococcus septicemia.

In seven cases in which old cavities had been secondarily infected with pyogenic cocci, these had caused a more or less general infection, although areas of acute mixed infection were not found in the lungs. In one of these cases (26) there was found general infection with streptococci, in another (19) a general infection with streptococci and pneumococci, in a third one (27) a general infection with pneumococci. One patient (32) had an acute pleurisy in which pneumococci and pseudo-diphtheria bacilli were found, another (16) acute pleurisy, acute pericarditis and a streptococcus septicemia, a third one (18) acute pleurisy, pericarditis, peritonitis, all due to streptococci, but no septicemia. In the last case (25) a purulent bronchitis with pneumococci and tubercle bacilli was found, and there were also some pneumococci in the spleen.

7. In two cases I have found what I believe to be the later stages of an infiltration of the pulmonary tissue with blood from aspiration during an attack of hæmoptysis. In both cases there were in the lower lobes quite extensive areas, which to the naked eye looked very much like areas of hæmorrhagic broncho-pneumonia. Sections showed the air spaces filled with red blood corpuscles. In a few places there were a few polymorphonuclear leucocytes, or a few epithelioid cells, and in the infundibula organisation of the blood had already commenced. In one case (5), in which such areas were found on both sides, a few tubercle bacilli could be discovered in the sections; in the other (6) no bacteria were found in the sections, but in a culture taken from the affected spot ten colonies of staphylococcus pyogenes aureus developed.

8. In two cases a chronic catarrhal pneumonia with beginning carnification was encountered. In case 30 it has not been possible to find any bacteria in the sections, but in an old cavity pseudo-diphtheria bacilli were present, and in case 39 one bunch of pseudo-diphtheria bacilli was found in one of the sections, and in a culture twenty colonies of pseudo-diphtheria bacilli developed.

Here, again, I should like to call attention to the coincidence of the presence of pseudo-diphtheria bacilli and carnification in cases 30, 36, 39.

Digitized by Google

In all my cases I have not been able to find one example of what Sata calls "käsige Mischpneumonie," that is, an acute mixed infection, with more or less extensive caseation in the middle of the affected areas. I have a suspicion that this may be due to climatic differences, because in a much shorter series of cases which I studied while in Göttingen, Germany, in Professor Orth's laboratory, I found four instances of this kind. I am confirmed in this suspicion by Spengler's observation that in certain climates—such as Davos, for instance—mixed infections seem to be much less frequent, and Spengler goes even so far as to ascribe the beneficial effect of mountain climate largely to the disappearance of mixed infection.

When one studies a series of cases of pulmonary phthisis at the post-mortem table, one soon gains the impression that in all cases there must be a long very chronic stage, which, if the patient does not die of some intercurrent disease, is terminated by an acute tubercular or mixed infection, which speedily destroys the life of the patient. This, however, is not the whole truth in the matter, as is abundantly proved by clinical experience. After a primary stage of slow, insidious development, most phthisical patients go through numerous periods of acute exacerbations and remissions. I believe that in all cases these exacerbations, as far as they affect the lungs themselves, are the result of a scattering of infectious material from pulmonary cavities by way of the bronchial tubes. The effect produced varies very much with the amount and the nature of the infectious material that is distributed through the lungs in this way. If the material contains a considerable number of pyogenic cocci, the immediate result will be an acute mixed infection, which, in course of time, may develop into a "käsige Mischpneumonie," or, as we have seen in case 36, into a more chronic condition with tendency to carnification. In many cases, however, especially as long as the patient is in a relatively good condition, the acute inflammatory process produced by the pyogenic cocci heals, and what becomes of the lesion afterwards depends very much on the number, and, perhaps, also the virulence, of the tubercle bacilli that remain. If they are few, a localised tubercular lesion will develop, usually in the infundibulum. If they are more numerous, the resulting lesion will have more the appearance of a tubercular pneumonia—chronic, sub-acute or acute, according to the number, distribution, and virulence of the bacilli. I have become more and more convinced that many lesions which, when examined at autopsy, present themselves as simple tubercular pneumonias, must start as mixed infections; and I am confirmed in this view by the result of some animal experiments which I made some years ago on guinea-pigs. At that time I was attempting to produce acute mixed infections in the lungs of these animals by injecting into one of the main bronchi, which I had catheterised, a mixture of pyogenic cocci and tubercle bacilli either from cultures or in the form of sputum from tuberculous patients that contained both. The experiments were not very numerous, but in all of them at the post-mortem the pyogenic cocci had disappeared entirely, and what I found was an acute tubercular pneumonia.

However, this is not true for all cases of tubercular pneumonia, certainly not

Digitized by Google

in this climate. As I have shown, it is not so very rarely that one here finds cavities containing tubercle bacilli in pure culture, and aspiration of a considerable amount of the contents of such cavities naturally would produce pneumonic complications which from the very beginning are purely tubercular (using the word in an ætiological sense). The possibility of such an occurrence is demonstrated in a most convincing manner in case 40.

The lesions started in one way or the other may progress to rapid caseation, and later the formation of many new cavities. Sometimes a secondary infection of such areas of tubercular pneumonia with pyogenic cocci from the cavities is observed.

On the other hand, we find evidence that often the acute changes slowly subside into more chronic ones, the acute tubercular pneumonia developing into a subacute one, and that into the more chronic stage, which in its turn may give way to more or less complete simple carnification (healing), as I have shown elsewhere,* or may also end in the formation of large fibrous tubercles (Sata).

In the large majority of cases it is only after a considerable number of such acute exacerbations and remissions that the pulmonary tissue becomes destroyed to such an extent, and the general health of the patient so much impaired that he succumbs to a renewed exacerbation of the diseased process in the lungs, either due to simple infection with tubercle bacilli or mixed infection, or to a more generalised terminal septic infection from the cavities.

In twenty of my thirty-nine cases, acute mixed infection, or sepsis originating from cavities that contained pyogenic cocci, seemed to be the immediate cause of death of the patient. Of the remaining nineteen cases, only in five (4, 36, 34, 1, 2) the death seemed to be directly due to the extent and character of the tubercular lesions in the lungs. In the other cases there were apparently other reasons for the death of the patient, which, however, with the exception of one or perhaps two instances—(5) acute articular rheumatism, endocarditis, and (29) arteriosclerotic contracted kidneys, and insufficiency of the hypertrophied and dilated heart)—had some connection with the diseased process in the lungs. In two cases (33 and 3) an acute infection of the pleura caused death, in one (13) the perforation of an intestinal ulcer. It is remarkable how frequently the giving out of a moderately hypertrophied heart (37, 24, 30, 14) seems to have been the last weight that tipped the balance over. In one case (38) there was a fatty heart and acute interstitial myocarditis. In the remaining four cases (23, 6, 39, 8) it is difficult to name a specific cause of death besides exhaustion, which in one case (23) was aggravated by profuse, uncontrollable diarrhœa, on account of extensive intestinal lesions.

All cases observed were in poor people, who had come to the City and County Hospital in San Francisco. I believe it would be of the greatest interest to study a similar series of consecutive cases under different social and climatic conditions.

---

* "Pneumonic Complications of Pulmonary Phthisis," *loc. cit.*

Digitized by Google

REFERENCES.

Orth: "Über Käsige Pneumonie." 'Virchow's Festschrift,' Berlin, 1891.

Ortner: "Die Lungentuberculose als Mischinfection." Wien und Leipzig, 1893.

Fränkel und Troje: "Über die pneumonische Form der acuten Lungentuberculose." 'Zeitschr. f. Klin. Medicin,' 1894, xxiv.

Spengler: "Ueber Lungentuberculose und bei ihr vorkommende Mischinfection." 'Zeitschrift f. Hygiene,' xviii., 2.

Schabad: "Mischinfection bei Lungentuberculose." 'Zeitschr. f. klin. Medicin,' xxxiii., 1897.

Schröder and Mennes, Bonn, 1898.

Sata: "Über die Bedeutung der Mischinfection bei Lungenschwindsucht." 'Ziegler's Beiträge.' 3rd Supplement, 1899.

Ophüls: "Pneumonic Complications in Pulmonary Phthisis." 'Amer. Journal of Medical Sciences,' July, 1900.

SYNOPSIS OF CASES EXAMINED.

All details that had no direct bearing on the question under consideration, such as the almost constant chronic miliary tuberculosis of liver, spleen, etc., and the frequent tubercular ulcerations in respiratory and intestinal tract, were omitted.

I. CASES WITHOUT MIXED INFECTION.

4.) I. 72. O. C. 32 years. Japan. Cook.

Development of disease.—Slight cough ten months before death, much worse and very hoarse three months later. Examined six months after onset, no physical signs, but many tubercle bacilli in sputum. Rapid progress; one month before death extensive dulness of apices. Since that time temperature rises to 104° in the evening. Confined to bed till death.

Post-mortem findings.—General atrophy. Many large old and more recent cavities, many conglomerated and disseminated miliary tubercles with simple hyperemia and oedema in their surroundings. In middle lobe a large area of tubercular (?) pneumonia with recent cavity.

Probable cause of death.—Extensive destruction of pulmonary tissue.

8.) I. 94. E. F.

Post-mortem findings.—Delicately built, well nourished. Small aorta and small bladder. Old cavities; disseminated and conglomerate old tubercles, moderately large areas of subacute tuberculous pneumonia with extensive caseation and recent cavities; circumscribed moderate recent tubercular pleurisy.

Probable cause of death.—?.

23.) III. 15. D. J. 32 years. California. Peddler.

Development of disease.—Cough began seven months before death. Rapid progress. Since fourteen days before death uncontrollable diarrhœa. No marked temperature.

Post-mortem findings.—General atrophy. Large old cavities and very large tubercular scars in both lungs. Coverslip from one of the cavities; a few tubercle bacilli, no other bacteria. (Tubercular ulcers of the intestine.)

Probable cause of death.—Exhaustion (diarrhœa).

37.) III. 51. W. McK. 26 years. Labourer.

Post-mortem findings.—General atrophy. Old cavities (one cavity pure culture of tubercle bacilli), chronic oedema near cavities, old conglomerate tubercles and areas of chronic tubercular pneumonia. Compensatory emphysema. Dilatation and hypertrophy of right ventricle.

Probable cause of death.—Insufficientia myocardii.

Digitized by Google

3.) I. 69. W. A.

Post-mortem findings.—General atrophy. Old cavities, many old disseminated and conglomerate miliary tubercles, small area of chronic tubercular pneumonia. Acute tubercular pleurisy on both sides, acute tubercular peritonitis.)

Probable cause of death.—Acute tuberculosis of serous membranes (pleuræ, peritoneum).

5.) I. 73. G. J. H. 25 years. England. Musician.

Development of disease.—Patient had been coughing off and on for two and a half years before his death. Symptoms severe for last half-year; died after contracting acute articular rheumatism.

Post-mortem findings.—Good state of general nutrition. Large old cavities, small tubercular scars; disseminated chronic miliary tuberculosis, blood in one of the cavities, old organised hæmorrhages containing a few tubercle bacilli in both lower lobes. Acute articular rheumatism, endocarditis mitralis recens.

Probable cause of death.—Acute rheumatism, endocarditis.

24.) III. 17. D. O'B. 36 years. Illinois. Boilermaker.

Patient gave no history of tuberculosis; was in hospital for a week with delirium tremens, and did not show any signs of phthisis.

Post-mortem findings.—General atrophy. Very old cavities (one cavity few tubercle bacilli in pure culture, another many tubercle bacilli, many pseudo-diphtheria bacilli), large tubercular scars, some old conglomerate tubercles, compensatory emphysema, simple hyperemia and oedema of left lower lobe. Dilatation and hypertrophy of right ventricle, fatty degeneration of heart muscle, general cyanosis.

Probable cause of death.—Insufficientia myocardii.

30.) III. 31. B. J. 33 years. Ireland. Labourer.

Development of disease.—Patient examined first three years three months before death. No dulness, no tubercle bacilli; emphysema. Next year hæmorrhage, beginning dulness at both apices, no tubercle bacilli; half a year later dulness increased. Almost one entire year with little or sometimes even no cough. At the end of this time increased area of dulness. Now rapid weakening, marked rises in temperature, delirium, exitus.

Post-mortem findings.—Strongly built, well nourished. Large old cavities (two cavities few tubercle bacilli, many pseudo-diphtheria bacilli), many old disseminated and conglomerate tubercles. Chronic catarrhal pneumonia with beginning carnification and considerable shrinking of left lower lobe (no bacteria either in culture or in sections), compensatory emphysema of right lung, hyperemia and oedema of left upper lobe, hypertrophy of right heart.

Probable cause of death.—Insufficientia myocardii.

33.) III. 40. D. J. 40 years. Hostler. California.

Development of disease.—First cough two and a half months before death. Claims to have lost 40 lbs. in three weeks. Very rapid development of disease in hospital.

Post-mortem findings.—Some atrophy. Large cavities, many large areas of chronic and subacute tubercular pneumonia with extensive caseation and softening. Pneumothorax and acute scrofibrinous pleurisy on left side, collapse of left lung.

Probable cause of death.—Pneumothorax.

40.) IV. 114. F. T. 36 years. Ohio. Bar-tender.

Development of disease.—Patient admitted to hospital the day before he died in a delirious condition. His friends state that he had been in bed for about three and a half months, and that he had been ill for some time previous, but they did not know just how long. When patient came to hospital he was apparently suffering from acute lobar pneumonia, both lungs being almost completely dull.

Digitized by Google

Temperature was 104° F. when he was admitted, and remained that high till a few hours before death, when it dropped to 102° F. He was delirious all the time.

Post-mortem findings.—General atrophy. Old scar with large central caseation and beginning softening (very many tubercle bacilli in broken-down parts, no other bacteria) in left apex. Old disseminated miliary tubercles in posterior parts of both lungs. Very acute tubercular pneumonia (very many tubercle bacilli in sections and coverslips, no other bacteria; of two cultures one was sterile, the other contained one colony of colonbacilli and one colony of staphylococcus pyogenes aureus) with caseation, and many small recent cavities involving both entire upper lobes and the entire middle lobe. Partial collapse and simple hyperemia and oedema (no bacteria in sections) of both lower lobes. Tubercular ulcers of tongue and epiglottis. Hypertrophy and dilatation of right ventricle.

Probable cause of death.—Acute tubercular pneumonia.

II. Cases with Mixed Infection, but in which the Mixed Infection seems to be of little importance.

A.—*Chronic Cases.*

14.) I. 114. S. D. 28 years. California. Waiter.

Development of disease.—According to patient's statement cough began two months before his death; four weeks before death both apices were found dull.

Post-mortem findings.—General atrophy. Large old cavities, many large conglomerate tubercles and areas of chronic tubercular pneumonia with beginning softening of caseous material (moderate number of tubercle bacilli, no other bacteria); in one bronchus signs of acute bronchitis (influenza-bac. (?) and tubercle bacilli); recurrent endocarditis mitralis, with stenosis, moderate hypertrophy of heart, cyanotic atrophy of liver, parenchymatous nephritis.

Probable cause of death.—Insufficientia myocardii.

6.) I. 84. M. C. H. 53 years. New York. Expressman.

Development of disease.—Patient claims to have been well till within three months before he died. Then he began to cough.

Post-mortem findings.—General atrophy. One very old cavity, many old conglomerate tubercles with beginning formation of cavities. In right lower lobe cone-shaped area of blood aspiration (no bacteria in sections, ten colonies of staphylococcus pyogenes aureus in culture); simple hyperemia and oedema. Cyanotic atrophy of liver (two colonies of staphylococcus pyogenes aureus), cystitis (colonbac.).

Probable cause of death.—?.

38.) III. 53. D. C. Woman, middle-aged.

Post-mortem findings.—General atrophy, anemia. Large old cavities surrounded by thick scars and chronic oedema, many old conglomerate tubercles, compensatory emphysema. In section of old cavity and in culture from lung a few pneumococci. Fatty heart. Acute interstitial myocarditis.

Probable cause of death.—Acute interstitial myocarditis.

36.) III. 47. Th. W. 61 years. Engineer.

Post-mortem findings.—General atrophy. Large old cavities (one cavity pure culture of tubercle bacilli), large tubercular scars, large areas of sub-acute and chronic tubercular pneumonia with extensive caseation and formation of cavities only tubercle bacilli), hæmorrhagic bronchopneumonia with many epithelioid cells and beginning carnification in left lower and middle lobes (in sections a few tubercle bacilli only, in culture twenty colonies of pseudo-diphtheria bacilli). Inflammatory enlargement and softening of spleen (spleen, kidney sterile). Liver fifteen colonies of streptococci.

Probable cause of death.—Extensive destruction of pulmonary tissue.

Digitized by Google

39.) III. 56. C. T. 39 years. Scotland. Shipbuilder.

Development of disease.—Patient has been subject to catarrh and frequent colds for a number of years. Confined to bed for about half a year before he died. Extensive dulness of lungs.

Post-mortem findings.—General atrophy. Old cavities. Large tubercular scars and old conglomerate tubercles, catarrhal pneumonia and carnifications of left lower lobe (in sections one group of pseudo-diphtheria bacilli, in culture ten colonies of them. No tubercle bacilli. In culture from liver fifteen colonies of pseudo-diphtheria bacilli).

Probable cause of death.—Chronic catarrhal pneumonia (?).

B.—*Chronic Case with Late Acute Development.*

34.) III. 42. S. W. 39 years. Washington. Bricklayer.

Development of disease.—Patient began to cough about five months before death. Rapid development, no remissions. No marked rise in temperature.

Post-mortem findings.—General atrophy. Old cavities. Many large old conglomerate tubercles with central cicatrisation and extensive caseation and softening in periphery, surrounded by areas of acute tubercular pneumonia (many tubercle bacilli, no other bacteria), compensatory emphysema, pneumothorax dexter, collapse of right lower lobe. In section one bronchus with purulent bronchitis (some tubercle bacilli, some pneumococci), parenchymatous degeneration o kidneys.

Probable cause of death.—Acute exacerbation of tuberculosis of lungs.

C.—*Acute Cases.*

1.) I 67. O. S. Chinese.

Post-mortem findings.—General atrophy. Old cavities. A few scattered old tubercles, extensive consolidations (acute and sub-acute tubercular pneumonia with many recent cavities, many tubercle bacilli, no other bacteria in sections, a few colonies of staphylococcus pyogenes aureus in culture from consolidated areas).

Probable cause of death.—Acute and sub-acute tubercular pneumonia.

13.) I. 113. N. A. 42 years. Norway. 'Longshoreman.

Development of disease.—Patient began to cough two years before death, and has been coughing ever since.

Post-mortem findings.—General atrophy. Old cavities. Many large areas of acute and sub-acute tubercular pneumonia with recent cavities. In one section small area of hæmorrhagic broncho-pneumonia (tubercle bacilli and pneumococci), tubercular ulcers of intestines, acute peritonitis, recent endocarditis, amyloid of spleen and liver.

Probable cause of death.—Perforated intestinal ulcer, acute peritonitis.

D.—*Acute Cases with Secondary Infection of Caseous Material.*

2.) I. 68. J. H. Old man.

Post-mortem findings.—General atrophy. Old cavities. Many large old conglomerate tubercles, recent very acute tubercular pneumonia with recent cavities. Recent cavities contain many staphylococci.

Probable cause of death.—Acute tubercular pneumonia.

29.) III. 30. K. W. Old man. Painter.

Post-mortem findings.—General atrophy. Old cavities, old conglomerate tubercles, some emphysema, sub-acute tubercular pneumonia in middle lobe with recent cavities with pneumococci in them, arteriosclerotic contracted kidneys. Marked dilatation of heart.

Probable cause of death.—Insufficientia myocardii.

Digitized by Google

III. CASES IN WHICH MIXED INFECTION PLAYS AN IMPORTANT RÔLE.

A.—*Tuberculosis with secondary infection and generalisation of infection.*

19.) II. 27. M. J. 29 years. California. Peddler.

Development of disease.—Patient began coughing about two years before death. At times he was almost entirely free from coughing. The last six months rapid and constant increase in symptoms. For last month patient has evening temperatures of 103° and 104° F.

Post-mortem findings.—General atrophy. Large old cavity. Many old disseminated and conglomerate miliary tubercles, compensatory emphysema, dilatation and hypertrophy of right ventricle, areas of chronic and one of very acute tuberculous pneumonia. In six old and more recent cavities pneumococci and pseudo-diphtheria bacilli. In cultures from spleen two colonies of pneumococci, thirty colonies of streptococci, liver and kidney sterile. Hæmorrhagic nephritis. Thrombi in heart and pelvic veins. Embolism in medium-sized branch of pulmonary artery.

Probable cause of death.—General infection with pneumococci and streptococci (?).

27.) III. 25. G. L. 17 years. Mexico. Tinsmith.

Development of disease.—First began to cough about 1½ years before death. Disease progressed continually without remissions.

Post-mortem findings.—General atrophy. Large old cavities (tubercle bacilli and pneumococci), large areas of acute tuberculous pneumonia with pneumococci in some of recent cavities. Empyema on both sides (tubercle bacilli and pneumococci), inflammatory enlargement and softening of spleen (pneumococci, pneumococci also in kidney), hyperemia and oedema (?) of left lower lobe.

Probable cause of death.—Pneumococcus infection.

32.) III. 37. T. W. J. 38 years. Massachusetts. Paperhanger.

Development of disease.—Patient began to cough about 2½ months before death. Disease progressed very rapidly.

Post-mortem findings.—General atrophy. Large old cavities (pneumococci and pseudo-diphtheria bacilli), old conglomerate tubercles, many large areas of subacute and chronic tubercular pneumonia with recent cavities, fibrinous pleurisy (pneumococci and pseudo-diphtheria bacilli), purulent pericarditis (sterile).

Probable cause of death.—Acute pleurisy.

16.) II. 1. C. F. 34 years. New York. Surveyor.

Development of disease.—Patient noticed first symptoms three months before he died, after exposure to severe chill. Disease progressed continually without remissions.

Post-mortem findings.—General atrophy. Old cavities (streptococci), scattered groups of old miliary tubercles. Patches of chronic tubercular pneumonia, capillary bronchitis and beginning catarrhal pneumonia (a few diplococci and tubercle bacilli) of right upper lobe. Pleuritis sero-fibrinosa sinistra, hydropericardium (streptococci), ascites chylosus (beginning tuberculosis), streptococcus septicemia, hemorrhagic nephritis.

Probable cause of death.—Streptococcus septicemia.

18.) II. 12. McP. A.

Post-mortem findings.—General atrophy. Very large old cavity, conglomerate tubercles and areas of chronic tuberculous pneumonia, large area of tubercular carnification with secondary streptococcus infection in spots and beginning formation of cavities, pleuritis sinistra (streptococci), pericarditis (streptococci), acute peritonitis. (Streptococci in liver, none in spleen and kidney).

Probable cause of death.—Generalised streptococcus infection.

Digitized by Google

25.) III. 19. McC. J. 36 years. Labourer.

Post-mortem findings.—General atrophy. Old cavities surrounded by large tubercular scars (two cavities tubercle bacilli and pneumococci, others tubercle bacilli in pure culture), large tubercular scars, old conglomerate tubercles, small areas of chronic tubercular pneumonia. In a recent cavity and in a bronchus with purulent bronchitis tubercle bacilli and pneumococci. Five colonies of pneumococci in culture from spleen, none in liver and kidney.

Probable cause of death.—Secondary infection with pneumococci.

26.) III. 20. Fl. J. P. 42 years. California. Swimming teacher.

Development of disease.—Cough began about five months before death. Examination four months before death, slightly diminished resonance in apices. Fourteen days later discharged, improved. Three days before death re-admitted in dying condition. Temperature 104° F. every night.

Post-mortem findings.—General atrophy. Old cavities (in one tubercle bacilli, pneumococci, a few staphylococci). Chronic fibroid tuberculosis with much shrinking of right lung. Near one of cavities (many tubercle bacilli) area of acute tubercular pneumonia, simple hyperemia and oedema of lower lobes. General infection with staphylococcus pyogenes aureus.

Probable cause of death.—General staphylococcus infection.

B.—*Tuberculosis of lungs with acute mixed infection.*

1. TUBERCULAR PROCESS ACUTE.

22.) III. 14. V. A. C. 50 years. Ohio. Barber.

Development of disease.—Patient claims that he was never sick till six weeks before his death. Four weeks before death extensive involvement of both apices was found clinically; course of disease very rapid; fourteen days before death patient delirious. Temperature every night 102–104° F. Last four days uncontrollable diarrhœa.

Post-mortem findings.—General atrophy. Old cavities (in three old cavities a few pneumococci, many tubercle bacilli), old miliary tubercles, large areas of subacute and acute tuberculous pneumonia; inflammatory oedema (some tubercle bacilli, pneumococci) of middle and both lower lobes.

Probable cause of death.—Acute mixed infection.

2. TUBERCULAR PROCESS CHRONIC.

15.) I. 121. C. B. 38 years. France. Baker.

Development of disease.—For a number of years patient has been subject to frequent heavy colds lasting usually about two weeks. They would come on always after some exposure. Last four and a half months before death coughed steadily. Last eight days delirious.

Post-mortem findings.—Moderately well nourished. Old cavities, large old tubercular scars, small area of acute tubercular pneumonia, patch of chronic tubercular pneumonia, inflammatory oedema (pneumococci, no tubercle bacilli) in middle lobe, catarrhal pneumonia (pneumococci and tubercle bacilli) in right upper lobe.

7.) I. 89. H. P. Old man.

Post-mortem findings.—General atrophy. Old cavities, old disseminated and conglomerate miliary tubercles, inflammatory oedema (streptococci, pneumococci; many tubercle bacilli) of middle lobe. Dilatation and hypertrophy of right ventricle, cyanotic atrophy of liver.

Probable cause of death.—Acute mixed infection.

9.) I. 98. K. R. 44 years. Germany. Razor-grinder.

Development of disease.—First began to cough about two years before death. Gradual but continual development. No rise in temperature before death.

Digitized by Google

Post-mortem findings.—General atrophy. Old cavities, large old conglomerate tubercles and patches of chronic tubercular pneumonia, acute broncho-pneumonia (many pneumococci, a few tubercle bacilli).

Probable cause of death.—Acute mixed infection.

10.) I. 104. H. B. H. W. 50 years. Germany. Clerk.

Development of disease.—Cough began two and a half months before death, Tapped several times.

Post-mortem findings.—General atrophy. Old cavities, old tubercular scars and conglomerate tubercles, hæmorrhagic broncho-pneumonia (very many streptococci, a few tubercle bacilli), purulent pericarditis (streptococci) hypertrophy of right ventricle, cyanotic atrophy of liver.

Probable cause of death.—Acute mixed infection.

17.) II. 4. R. E. F.

Post-mortem findings.—Moderately well nourished. Old cavities, old miliary conglomerate and disseminated tubercles, compensatory emphysema, inflammatory hæmorrhagic oedema of the left lower lobe (many pneumococci, non-identified bacilli, some tubercle bacilli), amyloid of spleen, kidneys, intestine, recent endocarditis.

Probable cause of death.—Acute mixed infection.

28.) III. 26. H. H. 46 years. American. Bar-tender.

Development of disease.—Patient had been coughing off and on for two years.

Post-mortem findings.—General atrophy, anemia. Old cavities, old disseminated and conglomerate miliary tubercles. Acute broncho-pneumonia in both lower lobes (a few tubercle bacilli, no cocci in sections, but colonies of pneumococci in cultures).

Probable cause of death.—Acute mixed infection.

3. LOCAL ACUTE MIXED INFECTION WITH GENERALISED INFECTION.

11.) I. 106. G. J. 47 years. Scotland. Carpenter.

Development of disease.—Symptoms began about half a year before death and progressed steadily. During the last week evening temperatures of 103° F.

Post-mortem findings.—General atrophy. Old cavity, old tubercular scars, chronic interstitial (syphilis (?) pneumonia, acute broncho-pneumonia (many tubercle bacilli, many streptococci), pleuritis, pericarditis, peritonitis, meningitis purulenta, streptococcus septicemia. Chronic syphilis, gout, amyloid.

Probable cause of death.—Streptococcus septicemia.

12.) I. 108. C. E. 26 years. California. Dish-washer.

Development of disease.—Patient began to cough five months before death. Was tapped for pleurisy. Temperature in last two months reached 102–103½° F.

Post-mortem findings.—General atrophy, anemia. Small old cavity. Scattered old miliary tubercles, small area of sub-acute tubercular pneumonia, inflammatory oedema (pneumococci and staphylococci in culture) of right lung. Staphylococci in liver and kidney, none in spleen. Acute tubercular pleurisy on both sides. Hypertrophy of right ventricle.

Probable cause of death.—Acute mixed infection; acute pleurisy.

20.) II. 45. R. J. 37 years. Portugal. Soapmaker.

Development of disease.—Patient began to cough a little over a year before death. At first the symptoms were very slight and not continuous. Six months before death physical signs were well marked. During last six months disease progressed slowly but steadily. About two weeks before death patient began to have an evening rise of temperature to 103–104° F., and at times was delirious at night.

Post-mortem findings.—General atrophy. Old cavities (in three, pure cultures of tubercle bacilli; in two, tubercle bacilli, staphylococci, streptococci), many old

Digitized by Google

conglomera tetubercles, inflammatory engorgement in both lower lobes (streptococci and tubercle bacilli). Streptococci in spleen, none in liver and kidney.

Probable cause of death.—Acute mixed infection.

31.) III. 36. W. G. N. 21 years. New Brunswick. Telegrapher.

Development of disease.—Patient first began to cough seven months before death. One month before death numerous patches of dulness scattered throughout both lungs. Since then disease progressed rapidly. For last week marked evening rise in temperature to 104–105° F.

Post-mortem findings.—General atrophy. Old cavities (many tubercle bacilli, few pneumococci), large tubercular scars, old conglomerate tubercles, areas of chronic tubercular pneumonia; acute broncho-pneumonia in left lower lobe. A few colonies of pneumococci in culture from liver, none in kidney and spleen.

Probable cause of death.—Acute mixed infection.

35.) III. 44. M. C. 24 years. Plumber.

Post-mortem findings.—General atrophy. Old cavities (in one pure culture of tubercle bacilli). Many large old conglomerate tubercles, areas of acute and sub-acute tubercular pneumonia with beginning carnification, oedema of left lower lobe (staphylococcus pyogenes aureus and streptococcus in culture). Streptococci and pneumococci in spleen, none in kidney and liver. Cyanotic atrophy of liver.

Probable cause of death.—Inflammatory oedema.

21.) III. 2. L. P. 28 years. Labourer.

Post-mortem findings.—General atrophy. Old cavities (in three pure cultures of tubercle bacilli), chronic disseminated miliary tuberculosis, some emphysema, large areas of hæmorrhagic broncho-pneumonia with beginning carnification in left upper and lower lobes (a few tubercle bacilli, in cultures a few pneumococci), inflammatory enlargement and softening of spleen. Fifty colonies of pneumococci in culture from liver, none in spleen and kidney.

Probable cause of death.—Mixed infection.

The CHAIRMAN: We are much indebted to these two gentlemen for their papers. I will ask Dr. Bulloch if he has any remarks to make.

Dr. BULLOCH, *London*: I have nothing fresh to say on the subject of mixed infection in tuberculosis, because I have not examined that subject specially, and that is due to the difficulties connected with it. I believe, from what I have seen of tubercular cases, that a great deal of erroneous knowledge is propagated with regard to mixed infection in tuberculosis. I believe if cases were carefully investigated by the clinical physician and afterwards by the skilled bacteriologist their researches would throw a great deal of light on the subject of mixed infections. It is not enough to simply see other bacilli present and then form theories on the matter, often unsupported by post-mortem evidence. The sputum should be examined during life, and after death preparations made from the lungs. Unless you are particularly interested in the case, and you have evidence of the particular part of the lung affected, you cannot make cultures, except with a definite object in view. But when there is careful co-operative work, such as I have indicated, very good results can be obtained. My own experience of tubercular sputum and the examination of lungs in mixed infections is that the results obtained by cultures are different from those in the case of the sputum. I think Mr. Foulerton said that an accurate estimate can be obtained by looking at this, but that is not my

Digitized by Google

experience. The question as to what these organisms do in the lungs, and to what extent they are passive and to what extent active, is very difficult to estimate. That is why I have not taken up the subject specially, but it is one which might very well be carried out by a combination on the part of the clinical physician and the pathologist.

The CHAIRMAN: It would be very interesting if the meeting would throw any light on the curious fact that in ulcerating tuberculosis of the lung and other parts of the body it is extremely rare to find any pyæmic abscess. I do not know that any satisfactory explanation has been given of that fact.

There is another matter, which I drew attention to in opening the discussion the other day, namely, the absence of a putrefactive odour in the tubercular lung. The lung of a tubercular person has a peculiar odour, but it is not exactly that of putrefaction, and yet we learn that proteus and other putrefactive organisms are present in the cavity. If we could get any information bearing upon that from the members of the Section to-day it would be a matter of very great interest.

I shall be glad to hear the remarks of anybody in the room.

Dr. FRIEDMANN, *Berlin*, read a paper entitled "On Experiments Proving the Infection of the Fœtus by Bacilli of Tuberculosis":—

MEINE HERREN,—Über Heredität der Tuberkulose ist schon viel geschrieben und gestritten worden. Trotzdem ist das bis jetzt vorliegende gesicherte Thatsachenmaterial ein ziemlich geringes.

Während die eine Gruppe von Autoren, an ihrer Spitze Robert Koch, nur eine Vererbung der Disposition annimmt, glauben viele andere hervorragende Autoren, so Baumgarten, Haupt, Liebermeister, Lugol, Landouzy, u.a., fest an einen direkten Übergang der Tuberkelbacillen selbst von den Eltern auf die Nachkommenschaft.

Eine solche Übertragung der Tuberkelbacillen auf die Frucht kann nun, wie *a priori* leicht ersichtlich, auf zweierlei Art zu Stande kommen, einmal von der Mutter aus auf dem Wege des Placentarkreislaufes oder aber durch sogenannte conceptionelle Infektion, d.h. direkt durch bacillenhaltiges Sperma.

Das Vorkommen einer tuberkulösen Infektion des Embryos durch den Placentarkreislauf ist sowohl für den Menschen als für die verschiedensten anderen Säugethierarten in einer grösseren Zahl von Fällen teils als zufälliger Befund teils experimentell mit Sicherheit constatirt worden. Auf die diesbezügliche ziemlich zahlreiche Litteratur will ich hier nicht näher eingehn, sondern möchte mir erlauben, hier auf meine ausführliche Publikation in der *Zeitschrift für klinische Medizin*, Bd. 43, Heft 1 und 2, zu verweisen.

Ganz anders aber steht es mit unseren Kenntnissen hinsichtlich der bei gesunden mütterlichen Organen durch den bacillenhaltigen Samen zu Stande kommenden tuberkulösen Infektion. Das Vorkommen dieses Modus der Infektion, eben der sogenannten conceptionellen Infektion, ist durch eine Reihe klinischer Beobachtungen bei Mensch und Thier in hohem Masse wahrscheinlich geworden, ja, sie erscheint der grossen Mehrzahl der erfahrenen

Digitized by Google

Aerzte heute wohl fast als unentbehrlich, aber sie ist bisher noch niemals sicher ewiesen worden. Man muss sich nämlich darüber klar sein, dass, um auch nur für einen einzigen Fall den exakten Beweis zu liefern, dass hier die tuberkulöse Infektion direkt vom Vater auf die Frucht übertragen ist, zweierlei erforderlich ist:

I. Der Nachweis, dass im Sperma des betreffenden Vaters virulente Tuberkelbacillen vorhanden sind;

II. Der einwandsfreie Nachweis, dass die Tuberkelbacillen in diesem Fall nur mit dem Samen und ohne jede Vermittelung der Mutter auf die Frucht übertragen sind.

Die unter I. genannte Vorbedingung, die eine bei weitem leichter zu entscheidende Frage betrifft, ist erfüllt, da eine grössere Anzahl von Autoren im Samen tuberkulöser Menschen und Thiere auch ohne Genitaltuberkulose virulente Tuberkelbacillen nachgewiesen haben.

Dagegen ist die Beantwortung der oben unter II. aufgestellten Frage bisher noch nicht gegeben worden, d.h. *der exakte Nachweis, dass gleichzeitig mit dem Samen in die Vagina gelangte Tuberkelbacillen ohne jede Vermittelung der Mutter direkt auf die Frucht übertragen werden, hat bisher gefehlt*, und hier, meine Herren, setzen meine Versuche ein, über deren erste Reihe ich Ihnen nun an der Hand einiger mikroskopischer Präparate, die Sie im Museum aufgestellt finden, in Kürze berichten möchte.

Ich bereitete mir eine sehr dünne Aufschwemmung von frisch gewachsenen virulenten Tuberkelbacillen in steriler leicht alkalisch gemachter Kochsalzlösung. Von dieser ganz leicht und gleichmässig getrübten Flüssigkeit wurden jedesmal einem Kaninchenweibchen im unmittelbarem Anschluss an die in meinem Beisein stattgehabte Begattung einige Tröpfchen in die Vagina injiciert. Die Spritze, die ich hierzu verwende, besitzt ein nach meiner Angabe hergestelltes, ungefähr der Länge des erigirten Kaninchenpenis entsprechendes Ansatzröhrchen. Untersucht man eine Stunde nach der Begattung das am äusseren Muttermund vorhandene Sekret, so findet man neben massenhaften Spermatozoen vereinzelte Tuberkelbacillen.

Die Mutterthiere wurden bisher regelmässig in den ersten acht Tagen der Gravidität (vom Tage der Begattung an gerechnet) getötet. Das meiste und beste Material stammt vom sechsten Tage. Ich verfüge über eine grössere Reihe sechs-tägiger Kaninchenembryonen, die ich sämtlich mit dem Uterus zusammen in lückenloser Schnittserie mikroskopisch untersucht habe.

Das Ei liegt bekanntlich in diesem frühen Stadium als eine zarte, dünnwandige, im Innern von Flüssigkeit ausgefüllte, aussen von der eng anliegenden Zona pellucida umschlossene Blase der Uterusschleimhaut erst ganz locker epithelial an: ein engerer organischer Zusammenhang zwischen mütterlichem und kindlichem Gewebe besteht nirgends, wie Sie sich mit einem Blick auf die im Museum befindlichen Präparate und Zeichnungen überzeugen können. Von einer Placenta ist in diesem frühen Stadium selbstverständlich noch keine Spur vorhanden.

In sämtlichen Embryonen von diesem Stadium fand ich nun zweifellose Tuberkelbacillen vor.

Digitized by Google

Die meisten Tuberkelbacillen finden sich in der embryonalen Zellschicht selbst, d.h. intracellulär. Doch sah ich auch mehrfach in dem minimalen, durch leichte Schrumpfung der Zellschicht zwischen dieser und der Zona pellucida entstandenen Hohlraum Tuberkelbacillen. Bei einem der Embryonen fand ich an einer Stelle in der embryonalen Zellschicht ein ganzes Nest von Tuberkelbacillen.

Die Organe der Mutterthiere erwiesen sich stets als gesund. Bei mikroskopischer Untersuchung fand ich niemals auch nur einen einzigen Tuberkelbacillus in der Schleimhaut des Uterus oder der Vagina.

Man gewinnt aus den Präparaten den Eindruck, als ob so gut wie alle mit dem Samen in die Vagina gelangten Tuberkelbacillen, die nicht in die Eier eingedrungen sind, aus dem Genitalkanal wieder eliminiert werden.

Die Gründe, aus denen für die Entscheidung unserer Frage zunächst die Untersuchung ganz junger Embryonen einzig und allein beweiskräftig und einwandsfrei war, liegen auf der Hand: sobald erst eine Placenta vorhanden ist, läge der Verdacht vor, dass die Bacillen durch diese, d.h. erst durch Vermittelung des mütterlichen Körpers, in die Frucht eingedrungen sind.

Nachdem jedoch durch meine Befunde an ganz jungen Embryonen die Eingangs gestellte Frage zum ersten Mal und im positiven Sinne entschieden war, habe ich eine grössere Anzahl meiner Mutterthiere austragen lassen. Über das weitere Schicksal der so gewonnenen neugeborenen Kaninchen, die als äusserlich normal entwickelte Thierchen zur Welt kommen, wie auch über die Ergebnisse von grösseren Reihen sich hier anschliessender Versuche werde ich später im Zusammenhang berichten. Dass meine oben beschriebene erste Versuchsanordnung den natürlichen Verhältnissen nicht entspricht, meine Herren, ist mir selbst wohl bekannt.

Dass aber Tuberkelbacillen, die mit dem Sperma in die Vagina gelangen, ohne jede Vermittelung der Mutter in die Embryonen übergehn, ist durch meine Versuche bewiesen.

The CHAIRMAN: Dr. Tatsusaburo Yabé, *Japan*, is not here, and we will take his paper as read.

That finishes the work of the Section, and I am sure we may congratulate ourselves on the very excellent papers which have been brought forward.

(The meeting then concluded.)

Digitized by Google

SUR L'IMMUNITÉ ET LA SÉROTHÉRAPIE DE LA TUBERCULOSE.

PAR TATSUSABURO YABÉ,

Médecin principal de la Marine Japonaise.

QUAND M. Koch a fait sa belle découverte de la tuberculine en 1890, il a annoncé l'existence de l'immunité de la tuberculose et il a trouvé l'influence exercée par l'infection tuberculeuse préexistante sur l'inoculation consécutive chez les cobayes. Chez les cobayes sains, l'inoculation tuberculeuse détermine la formation de l'ulcère typique qui persiste jusqu'à la mort de l'animal, mais si l'on pratique la même inoculation sur un cobaye déjà tuberculeux depuis quatre ou six semaines, on voit que ce point d'inoculation s'indure dès le deuxième jour et prend une coloration foncée. Bientôt se forme une véritable plaque de nécrose qui s'élimine, laissant une ulcération plane, laquelle se cicatrise habituellement très vite et guérit définitivement sans infection des ganglions lymphatiques correspondants. Cette découverte de M. Koch a été le premier rayon de lumière dans l'existence de l'immunité de la tuberculose. MM. Metschnikoff et E. Roux ont pu à plusieurs reprises confirmer cette influence de la maladie préexistante et ce phénomène typique a été appelé "Phénomène de Koch." Ils ont observé que l'infection ultérieure est toujours moins grave chez l'animal déjà tuberculeux et ils ont exposé que l'analogie avec les phénomènes connus pour les infections syphilitique ou morveuse, sous ce rapport, est indéniable. Ce phénomène de Koch bien affirmé, bien approfondi par MM. Metschnikoff et E. Roux, nous indique à coup sûr l'existence du mécanisme de l'immunité de la tuberculose, bien que ce soit à un très léger degré, et l'arrêt ou la guérison naturelle de cas prouvés par les anatomopathologistes fortifie encore cette hypothèse; mais malheureusement, toutes les expériences tentées dans ce but avec l'ancienne tuberculine n'ont donné aucun résultat. On mit alors en doute l'existence de l'immunité sur la tuberculose et on abandonna entièrement l'usage thérapeutique de la tuberculine.

M. Koch fabriqua par la suite un nouveau produit, la tuberculine résiduelle T. R. annonçant, grâce à sa nouvelle préparation, la possibilité d'immunisation de la tuberculose sur des cobayes, mais cette T. R. ne donna pas facilement sous la main d'autres chercheurs les résultats promis.

Des sérothérapeutistes prétendent à l'efficacité de leurs sérums, qui n'étaient que des sérums antituberculineux, en vérité, sans influence sur les bacilles tuberculeux, de sorte que ces sérums ne donnent aucun résultat positif dans les expériences sur des animaux de laboratoire, d'où les doutes sur l'existence même de l'immunité de la tuberculose.

Je me suis donné à la question de l'immunité de la tuberculose en 1898 dans le laboratoire de M. E. Roux à l'Institut Pasteur de Paris, et mon

Digitized by Google

premier but dans cette recherche était l'existence ou la non-existence de l'immunité.

Bien qu'on croie que la tuberculine soit la toxine principale des bacilles tuberculeux, elle n'en représente pas du tout l'action pathogénique, et elle n'a pas non plus d'action immunisante. Elle est seulement un réactif pour la tuberculose. J'ai donc cherché d'autres substances douées des propriétés pathogéniques des bacilles tuberculeux en conjecturant la faiblesse de la toxicité des toxines de ces bacilles; vu la grande différence qu'il y a entre l'action toxique des bacilles tuberculeux et celle d'autres microbes pathogéniques comme les bacilles du choléra, du tétanos et de la diphtérie. Dans le cas de la tuberculose, l'action toxique, locale, inflammatoire prédomine. Pour cette raison, je n'attache pas grande importance à la question de la dose mortelle à laquelle MM. Behring et Maragliano apportent tant d'attention. En même temps je partais d'un principe tout à fait différent de celui de M. Behring. Lui et ses élèves ont travaillé sur les toxines tuberculeuses pour y trouver des substances plus énergiques que la tuberculine à tuer des animaux sains et tuberculeux sans perdre le caractère de cette dernière. Mes expériences m'indiquèrent que la tuberculine n'a pas l'action des bacilles et qu'elle est un réactif pour la tuberculose. Donc, je me suis servi de la tuberculine plutôt pour déterminer le caractère des nouvelles toxines que j'ai analysées du corps des bacilles. Après avoir injecté d'abord une nouvelle toxine au cobaye sain je lui ai fait l'injection de la tuberculine quand il avait la température normale et j'ai observé la réaction fébrile.

Pour la fabrication des toxines, j'ai choisi les bacilles tués par la chaleur, parce qu'ils sont doués de la même propriété pathogénique que les vivants. Les bacilles morts sont justement la toxine brute ou naturelle, mais ils restent sans être absorbés dans le tissu injecté. Si on les décompose par des procédés chimiques, on peut changer les matières absorbables du tissu injecté, mais ces nouvelles substances perdront toujours leurs spécifiques actions pathogéniques; cependant il y a des substances qui peuvent représenter quelques propriétés des bacilles tuberculeux.

J'ai obtenu trois substances de corps bacillaires, en extrayant avec l'alcoo et l'éther, la solution des potasse et le réactif Schweitzer.

I.—La substance adipeuse, graisse ou plutôt cire (d'après Aronson);

II.—La substance protéique tuberculomycoprotéine;

III.—La substance chimiquement indéterminée appelée, de son action, tuberculobactéricidine, dont les caractères se rapprochent des nucléines.

Les deux premières substances suivies d'une injection de tuberculine donnent la réaction hyperthermique que ne donne pas la troisième. J'ai choisi la tuberculomycoprotéine et la tuberculobactéricidine pour mes essais d'immunisation, parce qu'elles représentent l'action essentielle des bacilles tuberculeux. L'une provoque une leucocytose au point d'injection et l'autre provoque la vascularité oblitérante, rôle capital pour la formation du tubercule, de même qu'elle produit la vascularité microscopique toxique sans bacilles, tout à fait semblable aux tubercules, type primitif de la méninge.

La tuberculomycoprotéine a été fabriquée par une submersion des corps

Digitized by Google

bacillaires dans une solution à 0,5 pour cent de potasse, précipitée par l'acide acétique, lavée par l'eau stérilisée, dissoute en 20 pour cent en volume dans de l'eau faiblement alcalinisée avec du bicarbonate de soude.

La fabrication de la tuberculobactéricidine a été faite par la submersion des corps bacillaires dans les réactifs Schweitzer. Le précipité formé par l'acide sulfurique, lavé par l'eau stérilisée, a été dissous à 10 pour cent dans l'eau alcalinisée avec du bicarbonate de soude.

Ces deux substances sont plus toxiques chez les cobayes tuberculeux que les cobayes sains. La tuberculomycoprotéine donne la réaction fébrique, tandis que la tuberculobactéricidine donne l'abaissement de la température avec une action toxique chez les cobayes tuberculeux, plus forte que la micoprotéine.

En essayant de donner l'immunité aux cobayes avec ces deux substances, j'ai aperçu que la tuberculobactéricidine peut déterminer le "Phénomène de Koch" et qu'elle donne l'immunité bactéricide. La tuberculomycoprotéine donne aussi l'immunité, mais plus difficilement que la tuberculobactéricidine, et j'ai pensé qu'elle donne plutôt l'immunité antitoxique, mon opinion étant que c'est une substance comparable à la toxine soluble d'autres microbes. D'après cela, j'ai conclu sur la question de l'immunité de la tuberculose comme suit (ce Mémoire a été publié à Paris, en novembre 1899).

I.—L'immunité de la tuberculose existe, mais elle est bien difficile à apercevoir d'un coup d'œil. Le phénomène de Koch est l'évidence de l'existence de l'immunité.

II.—La tuberculose est une maladie vaccinable et curable puisqu'on peut établir l'immunité contre elle par les injections de la tuberculomycoprotéine et de la tuberculobactéricidine.

III.—L'ancienne tuberculine ne représente aucune propriété des bacilles tuberculeux et elle n'a pas les propriétés pour être regardée comme une toxine *in vivo*, bien qu'elle nous embarrasse comme toxine principale par ses curieuses réactions. Ce n'est qu'un réactif pour la tuberculose et non plus une toxine. Elle n'a jamais l'action immunisante soit antitoxique, soit bactéricide. Quelquefois, elle amène l'influence favorable à la suite de sa réaction locale.

IV.—Les autres produits semblables à la tuberculine ancienne ont le même sort qu'elle.

V.—La nouvelle tuberculine T. R. est plus raisonnable que l'ancienne. C'est une substance tout à fait différente de par son mode de fabrication. Manquant d'expériences personnelles en cette matière, je réserve mon opinion, mais la méthode de sa fabrication me paraît peu raisonnable.

VI.—Toutes les substances nécessaires pour l'immunité restent dans le cadavre des bacilles tués par la chaleur.

VII.—La toxine tuberculeuse ne diffuse pas facilement, et elle reste locale là où siègent les bacilles.

VIII.—La mycoprotéine représente une action de leucocytose locale des bacilles tuberculeux et elle doit être vraisemblablement une toxine *in vivo*. Elle donne une immunité qui peut être regardée comme antitoxique.

IX.—La tuberculobactéricidine a une propriété pathogénique tout à fait

Digitized by Google

analogue à celle des bacilles tuberculeux. Quand on l'injecte dans l'organisme, elle manifeste très distinctement une action bactéricide. Elle donne une immunité bactéricide et peut être employée dans le traitement de la tuberculose.

X.—La mycoprotéine à chaud contient les deux substances. Elle est bien utilisable dans le traitement immunitif de la tuberculose en y ajoutant en plus une quantité de la tuberculobactéricidine.

XI.—La sérothérapie de la tuberculose est toute pleine d'espérances, mais l'immunisation doit s'exécuter par la tuberculomycoprotéine et la tuberculobactéricidine.

Mais je ne pense pas pratiquer l'injection de ces nouvelles matières aux malades comme M. Koch fait avec ses tuberculines ancienne et nouvelle. Pourquoi? Parce que ces deux substances sont bien toxiques chez les cobayes tuberculeux et même beaucoup plus toxiques chez eux que chez les animaux sains. Il y a grande difficulté à obtenir l'immunité chez les cobayes tuberculeux par ces injections. Leur toxicité dépend du degré de chaque cas. La tuberculomycoprotéine amène souvent la mort des animaux tuberculeux plus vivement que ne meurent les témoins qui ne reçoivent pas l'injection. La tuberculobactéricidine provoque toujours trop de sclérose; elle produit quelquefois une cirrhose du foie qui cause même l'ascite. Elle occasionne aussi de temps en temps la paraplégie. Quand on pratique l'injection des matières toxiques microbiennes aux malades pour leur assurer la guérison, on l'appelle toxinothérapie; mais cette toxinothérapie de la tuberculose par les produits des bacilles tuberculeux n'aura aucune valeur jusqu'à ce que la toxine injectée établisse l'immunité contre les actions pathogéniques de ce microbe. On comprend qu'on doive courir beaucoup de risques en pratiquant ce traitement aux malades qui sont d'autant plus sensibles à l'intoxication de ces toxines que leur cas est avancé.

On doit, cela va sans dire, obtenir un sérum antituberculeux pour l'usage thérapeutique. La deuxième partie de mes recherches était donc surtout de résoudre cette question.

Le sérum obtenu en injectant de la tuberculomycoprotéine et de la tuberculobactéricidine à des chèvres par mon principe montre une action bactéricide, mais il est bien pauvre en action antitoxique. Il s'ensuit que mon travail est encore bien imparfait au point de vue pratique, mais je ne le crois pas sans quelque intérêt au point de vue de la bactériologie pure, parce qu'on ne savait pas exactement jusqu'ici que les produits des bacilles tuberculeux donnaient un sérum bactéricide. Les sérums bactéricides contre les bacilles du choléra et de la fièvre typhoïde n'ont pas de valeur thérapeutique, il en sera de même avec le sérum bactéricide contre les bacilles tuberculeux, si le sérum n'augmente pas l'action antitoxique, bien que la tuberculose marche bien plus lentement que le choléra et la fièvre typhoïde.

Pour mesurer la valeur préventive du sérum contre les bacilles tuberculeux, il y a grande difficulté. Il faut bien connaître théoriquement la dose mortelle des bacilles tuberculeux, mais cela est bien difficile, car cette infection chez le cobaye est toujours fatale. La dose infectante doit être la dose mortelle,

Digitized by Google

mais l'animal survit trop longtemps en lui inoculant si peu de bacilles. Bien que la survie après l'inoculation soit bien différente, dépendant de la grosseur et de la résistance de l'individu, ainsi que de la virulence et de la dose de ces bacilles inoculés (l'inoculation de la même dose des bacilles tuberculeux est presque impossible en pratique, même si on broie avec le pilon un morceau de culture bacillaire dans le mortier), cette survie est la seule bonne indication en l'état actuel. La dose dont je me suis servi dans mon travail tue les cobayes de 350 à 450 grammes en deux mois et demi ou trois mois, rarement ceux-ci survivent cinq mois. A la première expérience de mon sérum, j'ai injecté trois grammes de mon sérum dans le péritoine de quatre cobayes et, le lendemain matin, j'ai inoculé une petite dose de culture bacillaire à la sous-cutanée en gardant deux témoins inoculés à la même dose. Pour continuer l'immunité, j'ai injecté trois grammes de sérum par semaine pendant trois semaines consécutives. Un témoin mourut après quatre mois cinq jours, et l'autre après cinq mois huit jours. Tous les quatre cobayes injectés de sérum jouissaient d'une excellente santé sans manifester de lésion tuberculeuse, mais deux succombèrent par suite d'un amaigrissement extraordinaire après quatre mois et sept jours et six mois seize jours. Les deux autres se portent toujours bien, même après un an et quatre mois. La deuxième expérience a été faite sur cinq cobayes avec deux témoins par le même procédé, mais en inoculant un peu plus de bacilles; les témoins moururent après trois mois dix-huit jours et quatre mois deux jours; quatre cobayes sont morts à la suite d'un amaigrissement tardif, qui commence vers le cinquième mois après l'inoculation. L'autre survit; ce cobaye survivant avait un abcès tuberculeux au point d'inoculation qui se développa vers le milieu du cinquième mois, après l'inoculation, et cet abcès guérit après deux injections de trois grammes de sérum sans infection générale. Il se porte bien maintenant. Les animaux qui moururent d'amaigrissement tardif, ne montrent pas de tuberculose à l'œil nu, les poumons sont indemnes, le foie est petit, de couleur bien foncée, noirâtre, la rate est petite et pâle. Sur les neuf cobayes de ces deux expériences trois ont donc survécu. J'ai ensuite essayé d'injecter le sérum à plus de trois grammes, mais ces essais ont échoué; le sérum venant à tuer les cobayes même sains presque toujours à quatre grammes, en quelques jours, en deux ou trois jours parfois. Ce sérum est toxique, en même temps il provoque souvent l'induration et même la nécrose, injecté à la sous-cutanée. L'inoculation dans le péritoine d'un mélange de sérum et de bacilles tuberculeux donne deux différents résultats. En certains cas, le péritoine reste indemne, mais les poumons sont atteints de tuberculose; en d'autres, une lésion du péritoine prédomine avec très peu de tuberculose dans les organes internes.

Les résultats de l'usage thérapeutique étaient les suivants. Dans les cas bien avancés, le sérum n'a aucune valeur pour sauver la vie des cobayes tuberculeux, ils meurent très souvent plus vite que les témoins. Si l'on injecte le sérum, l'amaigrissement survient. Les poumons sont atteints très souvent de l'hépatisation rouge, quelquefois même du lobe entier. La pleurésie avec épanchement n'est pas rare. Tous les tissus tuberculeux sont

Digitized by Google

sclérotiques. Si je commence la sérothérapie quelques jours après l'inoculation avec deux ou trois grammes de sérum en renouvelant l'injection tous les cinq ou sept jours, l'induration tuberculeuse au point d'inoculation reste stationnaire très longtemps en ne provoquant qu'un très léger gonflement des ganglions lymphatiques correspondants, et les cobayes survivent plus longtemps que les témoins, mais à la fin, ils succombent de tuberculose générale. Dans ces cas, la lésion tuberculeuse des poumons est gélatiniforme, le foie est gros et cirrhotique avec de gros tubercules, la rate est grossie aussi. Les examens microscopiques de ces tissus nous montrent très peu de bacilles.

Ainsi, mon sérum n'a pas l'efficacité suffisante ; mais ce qui, dans ce sérum, m'intéresse, c'est son action bactéricide. Injecté aux cobayes tuberculeux, il provoque la destruction des bacilles. Si l'on en injecte une certaine dose, cette action est bien visible en faisant les coupes microscopiques des tissus tuberculeux. Les bacilles disparaissent dans le tissu tuberculeux du foie et de la rate plus vite que dans celui des poumons. Une fois détruits, certains bacilles laissent souvent leurs parties granuleuses fortement colorées siéger à leur extrémité ou dans leurs lignes comme trace, dans les cellules où ils ont siégé (Préparation n° 1). En cas de destruction plus avancée on ne trouve presque plus de bacilles tuberculeux. Dans ces cas il y a des phagocytes qui contiennent des granules noirâtres. Ces granulations existent dans le foie et surtout dans les poumons (Preparations n^{os} 2 et 3). Il y a quelque difficulté à savoir si ces granulations sont des débris de corps bacillaires ou de poussière aspirées ou encore d'autres pigments. Même en admettant l'existence de poussière dans les cellules des poumons, il me semble que la plupart de ces granulations noirâtres proviennent de la destruction bacillaire parce que la même granulation existe dans le foie, mais c'est ce que je me propose de déterminer dans de futures recherches.

En examinant mes coupes, on admettra que mon sérum a une action bactérilytique sur les bacilles tuberculeux, mais ces coupes sont les tissus d'animaux morts de la tuberculose et la vie des cobayes n'est pas sauvée. La guérison de l'ulcère tuberculeux était annoncée comme signe de la guérison par M. Koch, mais la guérison de l'ulcère tuberculeux est plus facile que celle des tubercules des organes internes. La guérison de cet ulcère est obtenue très souvent par mon sérum, et quand l'abcès de la sous-cutanée crève, il se guérit bien vite, mais la tuberculose des organes internes persiste. L'évacuation des bacilles a de très favorables conséquences, comme nous le voyons sans cesse dans la clinique chirurgicale. Cependant, la guérison de l'ulcère est justement le signe d'augmentation de la résistance et elle n'est pas l'indication de la guérison tuberculeuse des organes internes. Ainsi on doit examiner toujours les organes internes. Jusqu'au point actuel de mes recherches, mon sérum n'a pas suffisamment d'action antitoxique, comme je vous l'ai dit, et je n'ai pas obtenu de résultats satisfaisants. A la suite de l'amaigrissement tardif des cobayes qui supportaient l'inoculation tuberculeuse, je songeai à me servir de bacilles vivants pour la fabrication des toxines et essayant d'une tuberculomycoprotéine fabriquée avec de ces bacilles, je trouvai qu'elle est beaucoup mieux que celle des bacilles tués par la chaleur. De plus, pensant

Digitized by Google

avoir perdu les toxines solubles qui amènent la cachexie en fabriquant avec des corps bacillaires tués ma mycoprotéine et ma bactéricidine, j'ai recommencé mes recherches avec une solution des bacilles vivants se décomposant dans l'eau chloroformée, mais ces recherches n'étant pas encore achevées, je ne saurais en donner les conséquences.

Conclusion.

L'immunité de la tuberculose existe, et le sérum bactéricide peut s'obtenir plus facilement qu'un sérum antitoxique, mais il ne suffit pas encore pour l'introduire en usage thérapeutique jusqu'au jour où on obtiendra le sérum aussi bien antitoxique.

Tel est actuellement l'état de mon travail, que j'ai pu poursuivre durant une partie de mon séjour à Paris, grâce au bienveillant accueil et aux bons conseils que je n'ai cessé de trouver en mon vénéré maître M. le Dr E. Roux, à qui j'adresse ici l'expression de ma profonde reconnaissance et de ma respectueuse estime.

Explication des Préparations.

N° I. Coupe des poumons d'un cobaye tuberculeux. Débris bacillaires détruits bien visibles dans le tubercule.

N° II. Coupe du poumon et du foie d'un cobaye tuberculeux ayant reçu en tout 8 grammes de sérum un mois et demi après l'inoculation. Les bacilles tuberculeux n'existent presque plus.

N° III. Coupe du poumon et du foie d'un cobaye tuberculeux ayant reçu en tout 12 grammes de sérum. Les bacilles tuberculeux disparaissent plus que N° II.

Toutes les préparations ont été fixées par l'alcool absolu, et colorées par la méthode de Ziehl.

A STUDY IN HEREDITY: IN ITS RELATION TO IMMUNITY AND SELECTIVE ACTIVITY IN TUBERCULOSIS.

By HERBERT MAXON KING, M.D. (New York University),
Grand Rapids, Michigan,

Delegate for the Michigan State Government to the British Congress on Tuberculosis, 1901.

The material for this study was taken chiefly from private practice, with the addition of a few cases occurring in the author's hospital service, whose opportunities for close observation at the time, and personal knowledge of the subsequent histories of the several cases after leaving the wards, were sufficient for the purposes desired. Some apology it would seem necessary to offer for presenting before such a body as this honourable Congress deductions and

Digitized by Google

conclusions based upon researches in so limited a number of cases as this present study includes, more especially since certain of the conclusions are at variance with traditional axioms in phthisiology. However, in the present inquiry all facts were collected and recorded personally and without prejudice, so that, however unique may be the conclusions to which a study of my cases has led, they are warrantable at least so far as the limit in number of observations will permit. The material at my disposal consists of carefully recorded histories of two hundred and forty-two cases of pulmonary tuberculosis. In every instance I have endeavoured, besides studying the influence of heredity and environment, to ascribe, whenever possible, a cause for such peculiar manifestations of the disease as became apparent, and also to establish the relation which certainly exists between the selective activity of the disease and hereditary influence.

The theory that a phthisical parentage predisposes the offspring to the disease by virtue of heredity, while very firmly grounded in the medical mind, and still more so, of course, in that of the laity, has nevertheless, I believe, always been a subject of more or less controversy, and especially since the isolation of the specific micro-organism. Without, however, entering upon a *résumé* of this controversy, which the limits of the present occasion and inclination alike prescribe, I have, like most phthisio-therapeutists, always been disposed to discredit the theory, and, until within the last four or five years, to place a negative value upon "family history" except in so far as a tuberculous parentage subjected an individual to additional risks of infection. The following instance, which came to my notice several years ago, first suggested to me that there might be a certain degree of *immunity* conferred on the offspring of phthisical parents. I published this instance as an illustration in 1898, in a paper in which I first ventured the opinion that "phthisical parents might impart to their progeny a certain immunity to the disease." * As it is brief and to the point I quote it:—"A clergyman and his wife, the latter having borne two children after developing pulmonary tuberculosis, took into their household by adoption the orphan child of a non-tubercular family. After surviving the birth of her last child six years, the wife died. Both of her children are living and in excellent health to-day, the younger having passed her twentieth year. The adopted child came under my observation at the age of seventeen years, having developed acute pulmonary tuberculosis, from which she died."

To adopt as a working theory this idea of conferred immunity in tuberculosis against all traditions requires, I admit, considerable temerity. One is constantly confronted with the question of inherited predisposition, and cannot fail to be influenced by the demonstrable fact of "percentage heredity," a term employed by Dr. J. Edward Squire to express in percentage the difference between the number of the offspring of consumptive parents subsequently developing tuberculosis, and that of the offspring of non-consumptive parents subsequently developing the disease. Dr. Squire's paper

* *New York Medical News*, December 3, 1898.

Digitized by Google

was published in November, 1897,* and was a most valuable contribution to the subject of hereditary influence in phthisis. In it he refers to a former paper, by himself, read before the Royal Medical and Chirurgical Society of London in December, 1894, on "The Influence of Heredity in Phthisis." Both papers were based upon results of thorough investigations in a sufficient number of instances to warrant the adoption of his conclusions as *standards* in future inquiry along this line. I have no hesitation in accepting them as final so far as they are carried.

This "percentage heredity" Dr. Squire found to be only 9·51, and he points out that it is easy to explain this small percentage of influence due to parents by the additional risks of infection to which the children of phthisical parents are exposed. Now, it has seemed to me almost certain that if these children of consumptive parents, especially that usually weak and under-nourished class from which Dr. Squire made his observations, were not in some manner protected, this percentage would be much larger, since in such cases we may take it for granted that all of them fulfil every required condition for the development of the disease. Therefore, while admitting the fact of a "percentage heredity," the very figures which express it are such as to support the theory of a hereditarily conferred immunity.

It is, I think, the rule to find among the children of consumptive parents poor nutrition, imperfect digestion, and other functional derangements, together with certain almost characteristic marks of degeneracy in the bony skeleton, the long, shallow and comparatively immobile chest, prominent scapulæ and sunken sternum. Yet with such features, more or less marked in a family of several children of consumptive parentage, it is a matter of common experience to see them all reach maturity without succumbing to an infection which, from the very nature of things, it would seem inevitable that they could not escape; while, on the other hand, the more acute, unresisting and progressively fatal cases, I think it is the common experience to find among those of a much more normal physical habit, and where the source of infection is frequently most difficult to locate. Observations of this character, however, prove nothing, of course. It is from the analysis of a given number of cases occurring under somewhat similar conditions, and among individuals of like hygienic and social relations, that statistics, to be of any value, must be drawn. Even so obtained, statistical figures are very apt to be misleading, and are no doubt open to much just criticism on that score. I would not attempt to prove a theory by conclusions based upon an experience in two hundred and forty-two cases unless the results were very positive, but I would very much like to provoke discussion of a subject which cannot be devoid of interest in such a society as this Congress represents, and to stimulate further inquiry into a matter of such far-reaching importance. If I shall have accomplished this object I shall rest content with the assurance that the theory will take care of itself.

To proceed, then; the cases which I have utilised in this series have

* "Heredity in Phthisis." *American Journal of Medical Sciences*, November, 1897.

Digitized by Google

occurred in a community for the most part moderately well-to-do—small tradesmen, mechanics, artisans, operatives in wholesome, well-lighted, clean and well-ventilated furniture factories, and small farmers. Nationality has been various. Next to American born, Dutch, German, Scandinavian and Irish predominated. Because of the comparatively recent development of the section of the country in which my observations have been made, and the more or less migratory habits of the first generation or so of foreigners in America, it has been the exception rather than the rule to be able to secure accurate data beyond the immediate parentage; but while it would be desirable to secure such data for purposes of demonstrating the transitory character of inherited immunity in tuberculosis, if such exists, for present requirements it is, I think, needless to do so.

The individuals comprising my series, by reason of their comparatively well-to-do condition and the customs prevailing in the locality, have none of them experienced the vicissitudes incident to life in the cheap tenement-house districts of larger cities. As a rule this class lives in small, compact, but well-lighted and comparatively new frame-houses, separated from each other by lawn spaces, and heated by stoves or occasionally hot-air furnaces. In the course of inquiry, when phthisis was found in either or both parents, it was the rule, with very few exceptions, to find it to have been present during the early life of the case under observation, and to have been the cause of death. Frequently it could be traced back prior to the birth of my patient, and to have persisted with remissions in activity for a considerable time afterward; more particularly was this the case among women, where in several instances I have satisfied myself of its presence during most of the child-bearing period. Without exception these instances occurred among women of consumptive parentage. Of course, positive demonstration of the long existence of the disease in these cases was obviously impossible beyond the time of my own acquaintance with the case, and as scientific evidence, therefore, such citations are valueless.

Of my 242 cases, the parents, one or both, had phthisis in sixty instances, or 24·8 per cent., while the parents were non-phthisical in 182 instances, or 75·2 per cent. Of the sixty who had phthisical parents, the father was found to have been affected in thirty-one cases, or 12·8 per cent.; the mother in twenty-nine cases, or 12 per cent.; and both parents to have been phthisical in four instances, or 1·6 per cent.

It was more common in my experience to find a previous tuberculosis in a brother or sister, or both, than in the parents, and in such cases an almost perfect history of infection could be traced. It was often possible to designate the date of infection to within a few weeks, and in some instances to within even narrower limits—a point of some value in estimating the period of incubation, or studying that questionable quantity, the "period of latency."

Seventy-four of my cases, or 30·6 per cent., gave a history of previous tuberculosis in a brother, sister, or both, and it is a point of some interest that of these seventy-four only twenty-two were of phthisical parentage; furthermore, as will be recalled, there were sixty cases in which one or both

Digitized by Google

parents were phthisical, and of these twenty-eight had brothers or sisters who were non-phthisical; or, to put it a little differently, out of sixty families with phthisis in one or both parents, there were twenty-eight instances of the disease affecting only one of several children. This does not, of course, imply that in the remaining thirty-two there was necessarily more than one child affected, for in many instances the patient was an only child.

It will further be seen that while twenty-two individuals having a phthisical parentage gave a history of previous tuberculosis in one or more brothers or sisters, there were fifty-two cases of non-phthisical parentage giving such a history.

These figures would certainly fail to show an hereditarily conferred predisposition to the disease, and, if they have any value at all, it is on the side of a conferred *immunity* instead. Of the whole number—242 cases—I can, at present, account for 235; of these 132 are living, and 103 have terminated fatally. Although it has a negative bearing upon the present inquiry, it may be of interest to note that in each group the percentage of phthisical parentage is the same. That is, of the 132 living, 26 per cent. had phthisical parents; and of the 103 dead, there were also 26 per cent. whose parents, one or both, had died of the disease.

The most interesting development in the course of this investigation has resulted from a study of the 103 fatal cases. As above suggested, 26 per cent., or twenty-seven cases, were found to be of phthisical parentage, while seventy-six had non-phthisical parents. My histories show that the *average* duration of the disease from its apparent incipience to the fatal termination was, in the incidence of the twenty-seven cases of tuberculous parentage, 4·01 years, while in that of the seventy-six cases of non-tuberculous parentage it was 2·93 years, a difference of more than a year in favour of those having a phthisical parentage.

Now, it has been my experience that the development of phthisis in individuals living or having lived for any length of time with consumptive parents, is more subtle and indeterminate as to commencement than is the case in the more robust offspring of non-consumptive families, where the onset of the disease is far more apt to be comparatively sudden, and consequently more easily determinable as to date of commencement. In this latter class the so-called "pre-tubercular stage" has no existence as a rule, and the measurement of the duration of the disease is accordingly much simplified. This "pre-tubercular stage" is, in reality, I am sure all will admit, a part of the life-history of the disease, and *should* be included with all subsequent stages of phthisis when estimating duration of the disease in a given case; but because I desired to free my inquiry as far as possible from misleading results or error in judgment as to what was and what was not the early stage of tuberculosis, I have, in this series of observations, limited the definition of phthisis to a stage of the disease where it has been clearly demonstrable. I have, therefore, a considerable leeway over and above the one year in favour of my cases with consumptive heredity, and had I included the "pre-tubercular" stage in my estimates I am very certain that the difference in duration

Digitized by Google

of the disease between the two classes would have been more marked than it is. I have, like many other observers, frequently demonstrated the presence of tubercle bacilli in the secretions of the air passages of healthy children and adults occupying apartments with consumptives, particularly consumptive parents, when subsequent events failed to show a development of tuberculosis from their presence. As an illustration: in a family consisting of a widow and four adult children, one of whom, a daughter, has been a consumptive for seven years (easily demonstrable during the whole of that time), the father had died after having had phthisis for upwards of twenty years. The consumptive daughter above referred to continued the local focus of infection after the death of the father, although to a less dangerous degree because of sanitary education. A few years ago one of the sons contracted typhoid fever, during the course of which the usual bronchitis developed. In the expectorated matter many tubercle bacilli were found during the course of the complication. Doubting the diagnosis of typhoid, I verified it by a positive Widal test and the diazo reaction in the urine. There was no pulmonary consolidation, and the case ran a typical course with good recovery, and, to my surprise, has never shown the least evidence of tuberculosis since, although during several subsequent winter attacks of bronchial catarrh a search has been made for bacilli. I might add that in this family every one of the four children is apparently a classical subject for phthisis, with the long, shallow and immobile chest and the imperfect nutrition above remarked; yet, though having lived almost their entire lives in the house with a consumptive, only one has developed the disease, and that one presenting every evidence of being able to successfully resist a fatal termination for many years to come.

Vital statistics show a gradually declining death-rate from phthisis. This is doubtless in part the result of improved methods in restricting the spread of the disease, but, in view of many facts, is no doubt also due in part to one or both of two not unimportant factors—*i.e.*, natural selection and inherited immunity. If the conclusions reached by Otto Naegeli, based upon 500 autopsies made in the Pathological Institute of Zurich, on bodies of individuals dying of various diseases, shall be verified in other parts of the world, the question of inherited immunity must be acknowledged to play a very essential part in the decline in frequency and severity of phthisis. Naegeli found indubitable evidence of active or latent tuberculous lesions in 97 per cent. of all sections in individuals between eighteen and thirty years of age, and in 99 per cent. of those over thirty years of age.*

If it is true that nearly all individuals reaching thirty years of age develop tuberculous lesions which are demonstrable *post mortem*, the only explanation for the comparatively small percentage who die from phthisis lies in that of an inherited or acquired immunity.

Within the memory of men still living there was no phthisis among the Indian tribes roaming the high plateaux of Colorado, Arizona, and New

* 'Virchow's Archiv.,' May, 1900.

Digitized by Google

Mexico. But a very few years after it became popular to send White consumptives to those regions as a therapeutic measure, the infection, thus spread among the Indians, gave rise to an almost epidemic form of phthisis of an unusually acute and fatal type. The same is true of the negroes of certain sections of the Southern States, whose heredity was entirely clean so far as tuberculosis is concerned, yet who succumbed in prodigious numbers as soon as the infection was brought them by the consumptive Whites from the North, who, by reason of their greater immunity, often recovered by virtue of a climatic influence which, like that of Mentone, was insufficient to protect the unimmunised native.

The older writers, in speaking of the influence of family history in phthisis, often remarked upon the frequency with which the disease affecting the grandparents would skip one generation, to reappear in the grandchildren. If there is truth in such a statement, it would seem plausible to attribute the phenomenon to an inherited immunity sufficient to protect the children, but not sufficiently permanent to protect the second generation. In my own experience I have not attempted records with this idea in view, and therefore have no facts to bear it out.

I must acknowledge one rather serious omission in the course of my investigations in the present series of cases. It was to a certain extent unavoidable, but in future inquiry along this line it seems to me most important that it be not again omitted if we are to arrive at any positive conclusions by such methods as I have employed. It lies in my failure to determine whether or not, when there was a consumptive parentage, the parent affected was tuberculous prior to the birth of the patient under consideration; for if such were not the case, and the parent became tuberculous only after the child was born, obviously any immunity to the disease which the child afterwards developed would be simply an acquired one; and if the child afterward became phthisical, the disease in the parent would have no more bearing upon the case than had it occurred in a brother or sister. In such a case it would be quite proper to include it in the class of non-phthisical parentage for the purposes of present study.

Briefly then, to summarise the results of observations in my series of cases, it will be remarked that:—

(*a*) Of 242 consecutive cases of phthisis, approximately one in every four gave a history of phthisis in the parents.

(*b*) Nearly one in three gave a history of previous phthisis in a brother, sister, or both.

(*c*) More than two-thirds of those giving a history of previous phthisis in brother, sister, or both had non-phthisical parents.

(*d*) As a rule, in the incidence of individuals of phthisical parentage afterwards developing phthisis, a much longer period was found to exist between the supposed exposure to infection and the subsequent appearance of the disease, than was the case in the incidence of those giving a non-phthisical heredity.

Digitized by Google

(*c*) Of 103 fatal cases of phthisis, the average length of life after development of the disease of those giving a history of phthisis in the parents, was to that of individuals of non-phthisical parentage approximately as 4 : 3.

So that, from a study of the foregoing series of cases, the following conclusions naturally follow :—

First, the percentage of consumptives having a tubercular parentage is actually smaller than that having a non-tubercular parentage, and much smaller than would be more than accounted for by the additional risk of infection to which the former class is subjected ; and

Second, tuberculosis in the parents renders to no inconsiderable extent an immunity to the disease in the offspring—an immunity which, of course, is but relative, and not sufficiently protective, but still demonstrable, as is shown by increased resistance to the progress of the disease and increased tendency to recover among this class.

ÉTUDE SUR QUELQUES TUBERCULOSES LOCALES.

FORMES CLINIQUES ET ANATOMIQUES PARTICULIÈRES DE LA LOCALISATION TUBERCULEUSE.

PAR M. DR. LE CLADO, *Chef des travaux de gynécologie à l'Hôtel-Dieu de Paris ; Délégué du Gouvernement grec.*

TUBERCULOSE RÉTRO-MALLÉOLAIRE.

CETTE forme de tuberculose mérite, par sa localisation précise, par la pathogénie et par des indications précises, de prendre rang parmi les formes cliniques de l'infection bacillaire. La description qui suit est basée sur trente cas environ recueillis depuis douze ans.

Avant ma première communication au Congrès de la Tuberculose tenu à Paris en 1891 (p. 786), aucun auteur n'en a fait mention. Depuis le professeur Duplay lui a consacré deux leçons cliniques (1895 et 1899).

L'infection bacillaire se localise dans une région anatomique bien définie, siégeant à la partie inférieure de la jambe au-dessus du calcanéum et derrière le tibia, au niveau de la jonction de la jambe avec le pied.

À ce niveau il existe un espace pyramidal dont la base est en bas et le sommet en haut. La base est représentée par la partie postérieure de la face inférieure—non articulaire—du calcanéum. La *paroi antérieure* est formée par la partie correspondante de la face postérieure du tibia tapissée par les tendons profonds de la jambe, qui y sont maintenus—dans leurs coulisses tendineuses respectives—par une forte aponévrose. La paroi postérieure est formée par le tendon d'Achille engainé aussi dans une aponévrose. Quant aux latérales elles sont formées par des feuillets aponé-

Digitized by Google

vrotiques tendues entre les bords du tendon d'Achille et les parties latérales de la paroi antérieure. Les feuillets font partie de l'aponévrose jambière qui entoure toute cette partie de la jambe, et va se continuer avec l'aponévrose du pied. Enfin le sommet résulte de la réunion des parois antérieures et postérieures. L'espace pyramidal ainsi limité contient du tissu graisseux abondant. C'est un véritable espace cellulo-graisseux, dans lequel se localise le tuberculôme s'y trouvant enserré dans ses parois pendant assez longtemps pour faire prendre et conserver à cette forme de la localisation tuberculeuse une physionomie à part, qui mérite la description qui j'ai donnée. Quant à son origine première, à son point de départ, nous le trouverons dans les organes qui limitent l'espace.

En bas la partie libre non-articulaire du calcanéum, de chaque côté des bords correspondants des malléoles, en avant la face postérieure de l'astragale. Autant de points osseux qui primitivement tuberculisés infectent l'espace celluleux rétro-malléolaire. Les bourses séreuses avoisinantes amènent aussi le même résultat. En première ligne celle du tendon d'Achille—bourse calcanéenne—dont la partie supérieure est libre dans l'espace celluleux, puis plus rarement, *beaucoup plus rarement*, les bourses des tendons profonds de la jambe appliqués contre le tibia de l'astragale.

En faisant agir l'hydrotomie sur cet espace j'ai pu séparer les différents plans aponévrotiques mieux que n'eût fait la dissection au scalpel.

Ces prémisses anatomiques posés pour l'intelligence du sujet, voici quelle est la définition que je donnerai à la maladie: *Une forme clinique d'infection tuberculeuse primitivement localisée dans l'espace rétro-malléolaire, souvent indépendante des synoviales tendineuses rétro-malléolaires et rétro-tibiales, et reconnaissant pour point de départ soit une altération tuberculeuse des malléoles ou du calcanéum, soit une tuberculisation de la bourse séreuse du tendon d'Achille.*

Sans doute l'espace rétro-malléolaire peut être envahi secondairement par une lésion tuberculeuse des articulations voisines ou des gaînes tendineuses, mais alors elle ne présente plus un type clinique spécial et ne mérite pas de rentrer dans ce cadre.

Je ne puis donner une description anatomo-pathologique précise, tout au moins pour les lésions à leur début, car dans les quelques cas qu'il m'a été donné de faire l'examen anatomique soit après autopsie, soit après amputation, les lésions étaient trop avancées, et des destructions étendues avaient eu le temps de se produire. Mais de faits cliniques m'autorisent à affirmer que cette forme de tuberculose existe en dehors de toute lésion articulaire ou tendineuse, ou de prendre les apparences d'une gomme tuberculeuse du tissu cellulaire déposée à froid dans l'espace rétro-malléolaire. Presque toujours un os, une pointe osseuse, en est l'agent principal ou bien la bourse du tendon d'Achille. Deux ou plusieurs points osseux peuvent être pris simultanément, voilà ce que l'exploration au stylet ou les opérations économiques m'ont appris.

Étiologie:—C'est une affection qui frappe l'âge adulte; du moins il ne m'a pas été donné de l'observer jusqu'à présent chez l'enfant. Elle est deux fois plus fréquente chez l'homme. On la rencontre avec une égale fréquence

Digitized by Google

aux deux membres, peut-être un peu plus souvent à droite. Comme dans toutes les tuberculoses on trouve souvent chez les malades des tares bacillaires personelles ou héréditaires — tuberculoses pulmonaire, pleurale, ganglionnaire antérieures au début de la localisation rétro-malléolaire; tuberculose chez les parents ou les collatéraux.

Les professions qui exigent un travail prolongé dans la station debout prédisposent manifestement à la tuberculose rétro-malléolaire (maîtres d'hôtel, cuisiniers, sergents de ville, hommes de ferme, femmes de chambre, etc.).

Comme cause occasionnelle j'ai relevé le traumatisme—contusion, entorse, grande fatigue bien antérieure à l'éclosion de la maladie. Chez deux de ces malades—femmes—la maladie a été précédée par une arthrite tibio-tarsienne d'origine génitale. En résumé, dans presque toutes les observations la localisation tuberculeuse aurait trouvé dans la région un *locus minoris resistentiae.*

Symptômes:—Le début est rarement aigu. Dans trois cas j'ai observé à la suite d'un traumatisme des douleurs violentes gênant la marche, et ayant pu faire croire à la production d'un phlegmon.

D'ordinaire le mal se développe insidieusement, rarement par une gêne douloureuse, presque toujours par le gonflement de la région, qui n'oblige pas néanmoins les malades à arrêter leurs occupations. Ceci est d'autant plus remarquable que le plus grand nombre de malades (vingt-cinq sur trente cas) exercent une profession les obligeant à travailler debout (maçons, camionneurs, mécaniciens, commissionnaires, maîtres d'hôtels, cuisiniers, etc.).

En somme, le premier signe qui apparaît est le gonflement. Il apparaît en dedans ou en dehors indifféremment derrière le malléole et au-dessous, sous la forme d'une saillie bien limitée en forme de bourrelet. Il faut noter que *ce gonflement bien localisé* peut être marqué par une tuméfaction générale de la partie inférieure de la jambe, conséquence d'une allure aiguë de la maladie, ou bien résultant de ce que le malade a marché pendant quelque temps. Le repos fait disparaître la tuméfaction générale en laissant ce gonflement localisé.

La maladie constituée se présente avec les caractères suivants: Derrière les deux malléoles, et au devant du tendon d'Achille, on remarque une tuméfaction allongée verticalement, *qui efface les deux dépressions rétro-malléolaires* et sur les côtés la saillie normale des bords du tendon d'Achille, faisant prendre à cette région une forme arrondie. Le gonflement ne remonte pas très haut, au plus six centimètres au dessus de l'espace rétro-malléolaire. En somme, en voyant une jambe ainsi malade la première idée qui se présente à l'esprit est que l'espace rétro-malléolaire est occupé par une collection anormale.

En général le gonflement est plus prononcé en dedans qu'en dehors. Il fait saillie de chaque côté, car en avant il est limité par les parties osseuses de la jambe—notamment les régions sus-malléolaires—et en arrière par le tendon d'Achille. En bas la conformation du talon ne change pas, et l'on voit la tuméfaction disparaître brusquement au niveau du calcanéum.

Les deux saillies latérales qui représentent le tuberculôme ne se prolongent pas en avant, en contournant les malléoles, comme le font certaines collections

Digitized by Google

tendineuses. Cette particularité s'explique par le fait que le tuberculôme rétro-malléolaire est bridé par les aponévroses, lesquelles empêchent toute contusion du tubercule en avant, tout au moins pendant un certain temps. Pour des raisons analogues les points des malléoles et le creux de la plante du pied conservent absolument leur apparence normale. La peau qui recouvre la région infectée conserve au début son indépendance et peut être isolée des tissus sous-jacents. Plus tard elle contracte des adhérences avec la partie fongueuse du tuberculôme et finit par s'ulcérer. Dès le début sa teinte est quelque peu altérée; sa couleur varie du rose pâle au rouge violacé. Dans cinq cas seulement la peau avait un aspect d'un blanc mat.

Cette coloration était intéressante à noter pour deux raisons: 1° Parce qu'elle contraste avec la décoloration habituelle de la peau qui recouvre les articulations tuberculeuses; 2° Parce qu'elle peut induire en erreur en faisant croire à la présence d'une collection suppurée, d'un vulgaire abcès. Dans quelques cas que j'ai observés (quatre cas), la tuméfaction prise pour un abcès profond a été incisée et de l'ouverture il n'est sorti que du séro-pus anguinolent et des fongosités.

Examen:—La tuméfaction est pâteuse aussi bien en dedans qu'en dehors, uniforme dans sa consistance; nulle part il n'y a des parties indurées ou franchement fluctuantes, et si ce caractère paraît quelquefois exister il n'est que trompeur. C'est une pseudo-fluctuation résultant non seulement de la présence des fongosités, mais du fait aussi du défaut de plan résistant pour l'immobilisation de la collection pendant l'examen.

Le meilleur moyen de rechercher cette pseudo-fluctuation et de se convaincre qu'il s'agit de fongosité ou de tissu carieux consiste à procéder comme il suit: Embrasser le talon avec la main en portant le pouce dans une des dépressions rétro-malléolaires, l'index et le médius dans l'autre, puis disposer les doigts de l'autre main, un peu au-dessus, à la limite supérieure de la collection en embrassant dans la concavité de la paume de la main l'extrémité inférieure de la jambe. Les doigts ainsi posés recherchent la fluctuation. On peut encore procéder à la même recherche en faisant placer la pulpe du pouce d'un aide dans la dépression rétro-malléolaire pour immobiliser le tuberculôme et servir de plan résistant, et en recherchant la fluctuation du côté opposé comme s'il s'agissait d'un abcès ordinaire.

D'un côté à l'autre la masse est mobile. On peut la déplacer facilement derrière les deux malléoles dans le sens transversal. Pendant ces manœuvres on ne provoque aucune douleur, quelquefois cependant l'exploration est très douloureuse.

Examen des parties voisines:—Le squelette de la région peut paraître cliniquement absolument normal; les malléoles et le talon se détachent bien, et ne présentent aucune modification, aucune augmentation de volume. Cet aspect est trompeur, et il faut alors chercher les points douloureux à la pression avant de se prononcer sur leur état en apparence normal.

Il est impossible d'explorer les gaînes tendineuses profondes autrement que par les mouvements des pieds sur la jambe. Dans la tuberculose rétro-malléolaire la flexion et l'extension du pied, même en opposant de la

Digitized by Google

résistance, sont indolentes. Il est évident que c'est là un signe diagnostique médiocre, mais je n'en connais pas de meilleur. Il y a bien aussi le gonflement profond, spécialement localisé le long du canal calcanéen, et du côté de la plante du pied, qui indique la tuberculisation des gaines profondes, mais alors la maladie est trop avancée et le diagnostic évident.

On peut isoler le tuberculôme rétro-malléolaire de la gaîne des péroniers latéraux et du tendon d'Achille. Pour se convaincre de l'indépendance et de l'intégrité de la première, il suffit de déplacer la masse fongueuse en arrière en insinuant la pulpe des doigts entre celle-ci et la gaîne des péroniers. Quant au tendon d'Achille il se dessine comme un ruban entre les deux bourrelets de la tuméfaction. Vue par sa face antérieure, la région du cou-de-pied est normale.

Les différents mouvements qui se passent dans l'articulation tibio-tarsienne, aussi bien que les mouvements de l'articulation calcanéo-astragalienne sont normaux et indolents. Pendant les mouvements il ne se produit pas de craquements. Les pressions les plus énergiques exercées de bas en haut sur le talon de manière à déterminer l'attrition des surfaces articulaires n'incommodent nullement les malades. Cette intégrité des articulations nous fait comprendre pourquoi les malades peuvent marcher pendant fort longtemps sans beaucoup souffrir de la présence du tuberculôme rétro-malléolaire.

Douleur :—Spontanée : elle peut se montrer même dès le début avec assez de violence pour compromettre la marche. D'ordinaire il s'agit plutôt d'une gêne se manifestant à l'occasion de la fatigue, ou bien des points douloureux localisés toujours sur un même point. À la longue, au bout de plusieurs mois, les souffrances obligent les malades à garder le repos.

La douleur provoquée, jointe à l'existence du gonflement, est une précieuse indice de lésion osseuse. Il faut toujours chercher à la provoquer en exerçant des pressions sur les parties osseuses accessibles ou sur la bourse calcanéenne. On explorera donc toute la partie libre du calcanéum, les faces cutanées et les bords des deux malléoles, et par des pressions profondes la face postérieure de l'astragale. Lorsqu'il existe une fistule elle conduit presqu'invariablement soit sur une surface osseuse dénudée, soit sur la bourse du tendon d'Achille.

Atrophie musculaire :—Je tiens à faire ressortir que l'atrophie du membre n'existe pas dans la tuberculose rétro-malléolaire, tout au moins dans les premiers mois de son existence. C'est encore là une indice d'absence de lésions articulaires.

État général :—Il reste d'ordinaire bon, les malades se nourrissent bien et conservent les apparences d'une bonne santé. Dans cinq cas d'autres lésions tuberculeuses existaient simultanément (pleurésie, tuberculose pulmonaire), et avaient déterminé un état cachectique.

Évolution :—Après une période incertaine, qui peut être comprise entre un mois et deux ans, caractérisée surtout par une légère douleur survenant après la fatigue, l'affection prend tout à coup des allures subaiguës. La tuméfaction s'établit ; elle prend la forme d'un abcès, et la collection s'ouvre à l'antérieur spontanément ou incisée par le chirurgien. La fistule s'établit

Digitized by Google

dès lors. Mais avec ou sans ouverture à l'antérieur, si l'on n'intervient pas, les gaînes tendineuses limitrophes, l'articulation tibio-tarsienne, et les articulations calcanéo-astragaliennes peuvent être envahies secondairement par l'infection, et l'envahissement des gaînes tendineuses profondes est capable de conduire le tuberculôme dans la plante du pied.

J'ai vu quatre fois l'aponévrose latérale de l'espace rétro-malléolaire céder, et la collection se répandre à la face cutanée de la malléole en contournant l'extrémité osseuse d'arrière en avant.

En résumé, il s'agit dans tous ces faits de tuberculoses secondaires pouvant infecter les gaînes tendineuses, les articulations ou bien le tissu cellulaire sous-cutané et la peau. Les différentes éventualités sont subordonnées à l'ouverture de la digue apronévrotique qui limite l'extension du tuberculôme. De ce fait le *pronostic* de la tuberculose rétro-malléolaire est grave à cause de la menace de propagation.

Diagnostic:—J'ai montré plus haut un certain nombre de signes devant servir au diagnostic différentiel. Je reviens sur certains points.

Lorsque la tuberculose affecte l'épiphyse postérieure du calcanéum, elle est caractérisée par un gonflement qui *déforme le talon*, et occupe les deux faces latérales de cette partie de l'os. Quelquefois la saillie talonnière est entourée d'un empâtement mal limité. Le pied se met en général dans l'équinisme, et il existe des points douloureux de chaque côté et à la face inférieure du calcanéum. La marche est difficile et douloureuse dès le début. Rien de semblable n'existe dans la tuberculose rétro-malléolaire.

Le diagnostic dans l'arthrite tuberculeuse des articulations astragalo-calcanéennes, et particulièrement de l'articulation postérieure, est plus délicat. Les seuls symptômes qui aient quelque valeur pour le diagnostic différentiel sont la difficulté de la marche dès le début à cause de la douleur et d'un certain degré de contracture; l'empâtement sous la malléole externe; les *mouvements d'abduction et d'adduction du pied qui, dès le début, sont difficiles, douloureux, ou même impossibles.* Ce sont là des signes délicats à bien mettre en évidence, aussi dois-je faire une réserve au point de vue de cette articulation, laquelle pourrait être en cause plus souvent que la clinique en saurait permettre de l'affirmer.

Traitement:—J'ai obtenu de bons résultats par ma méthode du chauffage bi-quotidien et l'immobilisation, au début de l'affection.

En cas de fistule le même traitement pourra être essayé en y joignant les injections modificatrices à travers la fistule, voir même la méthode sclérogène du professeur Lannelongue.

Plus tard, si l'affection persiste on doit traiter la tuberculose rétro-malléolaire comme tuberculose osseuse—ouverture, grattage des fongosités, résections partielles ou simplement abrasion à la curette portant sur les os malades, etc. Si les lésions sont très étendues, il ne faudrait pas hésiter à pratiquer une large opération en enlevant au besoin la totalité de l'astragale et une partie du calcanéum. Dans toutes ces interventions la cautérisation ignée trouve une indication.

Digitized by Google

Tuberculose Partielle de la Trompe.

J'ai observé chez la femme cinq cas de tuberculose interstitielle et ulcéreuse (forme mixte) qui méritent d'attirer l'attention. Ces cas peuvent être divisés de la façon suivante.

(*a*) *Première catégorie*, comprenant deux cas dans lesquels la portion *cylindrique de la trompe* dans *sa partie initiale*, tout près de l'angle utérin, était occupée par un nodule tuberculeux, isolé, encerclant et absorbant entièrement cette partie de la trompe. Le volume de ce tuberculôme était environ celui d'une petite noisette. Le reste de la trompe était sain et perméable, mais au niveau de la lésion le canal était entièrement fermé. La variété histologique repondait à la structure dite forme mixte. Sur ce point les tuniques de la trompe, à part le péritoine piqué de granulations grises, étaient absolument méconnaissables; le tissu pathologique avait remplacé le tissu normal. Les deux sujets ne présentaient nulle part ailleurs des lésions tuberculeuses cliniquement appréciables. Cette lésion mérite d'être rapprochée des fibromes qui siègent sur la même portion de la trompe, et qui y restent strictement localisés. Je pense qu'elle doit se développer autour de l'anastomion, ou des branches anastomotiques, de l'utérine et de l'utéro-ovarienne, peut être au dépens des lymphatiques si abondants en ce point. Je ne puis dire s'il s'agit d'une lésion primitive, ou si l'entrée de l'infection tuberculeuse se trouve sur un autre point des organes génitaux de la femme.

(*b*) *Deuxième catégorie*, comprenant trois cas. Il s'agit encore d'un tuberculôme interstitiel, mais la lésion est moins strictement localisée, un peu plus diffuse. Elle occupe l'angle de l'utérus et la portion initiale de la trompe. Deux fois la lésion était assez profonde pour s'avancer à un centimètre de profondeur dans l'épaisseur de l'angle utérin, ce qui a nécessité une résection de presque tout l'angle utérin. Dans le troisième cas la lésion était plus superficielle. Le tuberculôme m'a paru dans les trois cas avoir pris naissance dans la cavité de la trompe pour envahir ensuite toutes ses tuniques et la paroi correspondante de l'angle utérin. Sur le pourtour de la lésion à la surface péritonéale de l'utérus, et sur la partie correspondante de la trompe, il existait un semis de granulations grises. Cette lésion diffère de la précédente par la présence à sa superficie d'une ulcération. C'est un tuberculôme ramolli et ulcéré, mais par son siège, par ses relations avec la trompe, par sa structure histologique, elle paraît être identique à la précédente.

Dans deux cas l'intestin adhérait à l'ulcération, et à son tour il présentait des granulations tuberculeuses sur une petite étendue de sa surface péritonéale. Il s'agit, sans doute, d'une tuberculose secondaire par contamination directe, ayant atteint l'oméga dans un cas, l'intestin grêle dans l'autre. Dans le troisième fait il n'y avait pas d'adhérence intestinale, et nulle part ailleurs je n'ai trouvé, dans le ventre, des granulations tuberculeuses. La faible adhérence de l'intestin, dans les deux cas, le peu d'étendue en profondeur des lésions intestinales, l'absence complète de manifestations cliniques du côté des voies digestives, prouvent que la contamination de l'intestin est secondaire, et que ce n'est pas cet organe qui a infecté l'angle de l'utérus et la trompe.

Digitized by Google

Après extirpation la lésion se trouve être constituée identiquement comme les précédents, par un tuberculôme interstitielle ulcéré à la surface. La trompe a perdu sa structure normale, mais la partie correspondante de l'utérus semble mieux résister.

Au point de vue clinique les données ne sont pas assez significatives pour conduire à un diagnostic précis. Dans ces cinq cas trois fois il fut impossible de trouver à l'exploration clinique aucune lésion pouvant être attribuée à la tuberculose. Dans un cas j'ai retrouvé dans le passé de la malade, dans son enfance, des manifestations de scrofulose—maux d'oreilles, yeux, gourme, suppurations ganglionnaires—la malade portait même encore de cicatrices au cou. Dans le dernier cas enfin la scrofulose infantile était également indéniable, mais il existait aussi des lésions tuberculeuses aux deux sommets, n'ayant pas dépassé la deuxième période. Dans l'ensemble l'état général paraît se conserver bon et assez longtemps.

Les symptômes n'ont rien de précis, mais les troubles fonctionnels ont toujours été assez prononcés pour me décider à intervenir chirurgicalement.

Ce sont des symptômes d'une ventro-salpingite, dont le phénomène clinique dominant est la douleur.

Du côté de la matrice les phénomènes mètritiques ont été dans deux cas seulement très accusés et très persistants. Une des malades même a dû être curettée à deux reprises. Dans trois cas les douleurs abdominales étaient intolérables, et dans les deux autres toujours assez fortes pour constituer une indication à l'intervention chirurgicalement. À l'exploration on arrive aisément à sentir le nodule, qui est très douloureux à la pression du doigt. Chez l'une des cinq malades il existait assez de sensibilité pour nécessiter l'emploi du chloroforme en vue de l'examen clinique.

La lésion paraît rester longtemps localisée; autant qu'on put juger par l'interrogatoire dans les trois cas où la lésion était ulcérée, la durée n'a pas été plus courte que dans les deux autres. En moyenne les premiers symptômes remontent à deux ans auparavant. Le pronostic paraît donc d'autant meilleur que quatre fois sur cinq la lésion était unilatérale.

Le seul traitement qui convient me paraît être l'ablation avec l'instrument tranchant. La cautérisation ne me paraît pas de mise. Toute la trompe doit être sacrifiée, et au besoin on résignera une partie de l'angle utérin. On doit entraver les parties cruentées et refermer le ventre. Sur cinq cas j'ai eu en opérant ainsi cinq succès, sans récidive (le plus ancien remonte à six ans).

En terminant je dois de nouveau faire ressortir le rapprochement qu'il convient de faire entre la tuberculose angulaire de la trompe et les fibromes ayant le même siège. Les deux lésions, en effet, présentent le même siège, et se comportent vis-à-vis des parois salpingiennes de la même façon. Cliniquement elles présentent un même symptôme dominant, la douleur, qui, dans les deux cas, constitue une indication à l'intervention chirurgicale.

Il est possible que cette localisation spéciale du tuberculôme soit sous la dépendance de l'anastomose de l'utérine et de l'utéro-ovarienne, pour sa localisation, et du tissu lymphatique pour son développement.

Digitized by Google

Tuberculose Hémilatérale.

Au Congrès de la Tuberculose, tenu à Paris en 1891 (p. 324), j'ai communiqué six observations de nature à faire admettre que la tuberculose peut se localiser pendant une longue période d'années (cinq à dix ans) dans une moitié du corps.

Depuis cette époque il m'a été donné d'observer encore sept cas qui mettent hors de doute cette intéressante particularité.

L'analyse de ces observations montre: 1° Que la diathèse héréditaire ou acquise n'exerce aucune influence sur le développement de *la tuberculose hémilatérale.*

2° Que le côté droit ou gauche du corps peut être indifféremment infecté.

3° Qu'un affaiblissement de la nutrition de la moitié du corps peut constituer un appel à cette localisation. Dans un cas, en effet, il s'agissait d'une jeune fille de dix-huit ans ayant été atteinte de paralysie infantile du membre inférieur gauche; et dans un autre cas il y eut du même côté, chez une femme âgée de quarante-cinq ans, préalablement et à plusieurs reprises des éruptions de zona.

4° Que les points infectés par le bacille peuvent n'avoir aucune relation lymphatique directe ou, tout au moins, que l'infection ne suit pas le cours de la lymphe pour se localiser successivement sur tel ou tel point.

5° Que les points sur lesquels la tuberculose s'est localisée sont: Les articulations—coude, poignet, genoux, tibio-tarsienne, etc.; les ganglions du cou et de l'aine, ceux de l'aisselle; le testicule; la plèvre; les gaines tendineuses—des doigts, de la main, des péroniers et extenseurs du pied; les côtes, les métatarsiens, la bourse séreuse périthoracique de Verneuil; la mamelle; enfin, la peau—lupus de la face et de l'aile du nez.

6° Qu'à partir de la première localisation, chacune de celles qui suit peut apparaître immédiatement à la suite de celle qui précède, ou mettre un espace de temps fort long—une trève de plusieurs années même.

7° Qu'il n'est pas nécessaire que la localisation précédente soit en pleine évolution pour voir apparaître une nouvelle attaque. Dans la moitié des cas environ, c'est après la guérison par une opération, ou par un traitement médical, que la nouvelle infection se fait. Il y a donc véritable récidive.

Quelques exemples feront comprendre ce qu'il faut entendre par tuberculose hémilatérale.

I. *Jeune femme de vingt-huit ans; observé à l'Hôtel-Dieu en* 1890. Tuberculose de la mamelle; amputation; curage de l'aisselle; guérison. Elle rentre, à la fin de 1891, avec une arthrite tuberculeuse du genou. Les deux localisations affectaient le côté droit.

II. *Homme de trente-cinq ans* (*Hôtel-Dieu*, 1890):—Amputé, à la Pitié trois ans auparavant, du testicule gauche pour une arthrite tuberculeuse. Il entre à l'hôpital avec une tuberculose tarso-métatarsienne gauche et une adénite tuberculeuse du cou, même côté.

III. *Hôtel-Dieu*, 1894:—Femme de vingt-cinq ans. Dix ans auparavant pleurésie droite guérie. Cinq ans après arthrite tuberculeuse du coude guérit

Digitized by Google

par ankylose, côté droit. En 1894 tumeur blanche du genou droit avec infection des ganglions de l'aine du même côté.

IV. *Hôtel-Dieu*, 1900 :—Jeune homme de dix-huit ans. Arthrite tuberculeuse du coude droit depuis six mois, sans fistule. Depuis un mois synovite des gaînes pulmonaires du côté droit, adénite cervicale.

V. Je citerai aussi un cas d'une jeune femme de trente-trois ans, que j'ai observé, avec mon regretté maître Verneuil, en 1888 à l'Hôpital de la Pitié, qui a présenté cinq différentes localisations dans l'espace de douze ans, siégeant toutes du même côté, à gauche : Pleurésie, tuberculose des ganglions du cou et de l'aisselle, spina-ventosa du médius, arthrite tuberculeuse tibio-tarsienne.

Dans d'autres observations l'infection remonte, suivant le cours de la lymphe, de bas en haut.

Cette localisation de l'infection tuberculeuse comparable à l'œdème hémilatérale (Potain), à l'hypertrophie hémilatérale (Trelat, Monod), à certaines éruptions à répétition, tend à confirmer l'individualisation pathologique possible d'une moitié du corps à l'exclusion de l'autre.

Il faut voir dans ces faits une action (probable) du système nerveux.

Comparativement à la fréquence énorme des tuberculoses locales, le nombre des faits que j'apporte pourrait être considéré comme une sorte de coïncidence. Tel n'est pas mon avis. Ne suffit-il pas pour s'en convaincre de prendre en considération ces observations "principes," où plusieurs localisations attaquent successivement une même moitié du corps en respectant l'autre, sans que la fatigue ou le traumatisme puissent être invoqués dans l'étiologie ? Je suis certain que, l'attention des membres du Congrès attirée sur ce point, le nombre de cas ultérieurs ne tardera pas à augmenter, et que ce point intéressant de l'infection tuberculeuse sera mieux étudié.

EFFECT OF EXPOSURE TO LIQUID AIR UPON THE VITALITY AND VIRULENCE OF THE BACILLUS TUBERCULOSIS.

By HAROLD SWITHINBANK.

A SERIES of experiments carried out early in the year 1900, with the object of testing the effect of the temperature of liquid air upon the vitality and virulence of the bacillus tuberculosis, produced results which—although in complete accord, as far as the question of vitality was concerned, with those arrived at by Professor McFadyean in the carefully planned experiments reported to the Royal Society on the 1st of February and the 5th of April, 1900—raised some doubt in my mind as to whether the abnormally low temperature, continued for a lengthened period, might not have some modifying effect upon the virulence of the organism. I decided, therefore, in the month of January of this year, to put the question to the test of an experiment which I hoped would be conclusive.

Digitized by Google

The questions to be solved appeared to me to be :—

1. Whether exposure for varying periods to the temperature of liquid air had any effect upon the vitality of the bacillus tuberculosis.

2. Whether such exposure in any way modified its virulence.

3. Whether time was a factor in the question.

4. Whether, as is the case at the higher end of the thermometric scale, successive alternations of temperature had any special effect.

5. Whether actual contact * with liquid air, if obtainable, produced any special results.

The experiments, which were carried out in duplicate, lasted over a period of five months, and I am greatly indebted to Dr. Debrand, of the Pasteur Institute, not only for his general supervision of the experimental animals, but also for his kindness in making the autopsy of one complete series as a control.

A special strain of tubercle, isolated from a human cervical gland, was used for the purpose of inoculation. Sub-cultures of this were made upon potato, and the *raclage* from these was used throughout the whole series of experiments. This was enclosed in specially made tubes, and submitted to the influence of liquid air as follows :— †

Tubes A. Six hours' continuous exposure to liquid air, without contact.

Tubes B. Twelve hours' exposure, without contact.

Tubes C. Twenty-four hours' exposure, without contact.

Tubes D. Twenty-four hours' exposure *with contact*, the tubes remaining filled with liquid air during the whole period.

Tubes E. Forty-eight hours' exposure, without contact.

Tubes F. One hundred and forty-four hours' exposure, without contact.

Tubes G. One week's exposure, without contact.

Tubes H. Six weeks' exposure, without contact.

Tubes K. Six weeks' exposure *with contact*, the tubes remaining filled with liquid air during the whole period.

To test the question of successive alternations of temperature :—

Tubes L. Six alternate exposures of one hour each during twelve hours to the temperature of liquid air and that of 15° C.

Tubes M. Three alternate exposures as above, followed by six hours' continuous exposure to liquid air.

Tubes O. Controls.

---

* The word "contact" is used throughout, but it is doubtful whether actual *contact* is possible. Given that a cell contains a large proportion of water, it is questionable whether the admission of liquid air to the tube containing the organisms would not give rise to the immediate formation around each individual cell of a thin coating of ice, which would effectually protect the cell contents from any specific action the liquid air might possibly have upon them.

† The temperature of liquid air may be taken at − 193° C., the actual temperature to which the organisms were exposed as − 186° C.

Digitized by Google

The effect of the above treatment, judged by the result of the subcutaneous inoculation of the guinea-pig with an emulsion made from the contents of one of each series of the above tubes, will be shown by the following Table. Thirty animals in all were inoculated, and one-half cc. of the emulsion was used in each case. A full *précis* of the post-mortem results will be found in the "Proceedings of the Royal Society" for 1901.

The question of vitality was tested by making sub-cultures from the tubes after exposure. With the exception of those tubes exposed to alternations of temperature, no difficulty was found in obtaining a luxuriant growth. Exhibited in the Congress Museum will be found (1) a control culture before exposure, and (2) sub-cultures on potato from tubes exposed, with and without contact, for a continuous period of forty-two days.

RESULTS OF SUBCUTANEOUS INOCULATION INTO THE GUINEA-PIG OF ONE-HALF CUBIC CENTIMETRE OF AN EMULSION IN BROTH OF CONTENTS OF TUBES TREATED AS ABOVE.

Tubes.	Treatment.	Animal died or killed.	After.	Précis of Post-mortem results.
A.	6 hours without contact	Killed	100 days	Tubercular.
B.	12 hours without contact	Killed	100 days	Tubercular.
C.	24 hours without contact	Died	95 days	Tubercular.
D.	24 hours *with contact*	Killed	100 days	Tubercular.
E.	48 hours without contact	Killed	200 days	Tubercular.
F.	144 hours	Died	78 days	Tubercular.
G.	1 week without contact	Died	97 days	Tubercular.
H.	42 days without contact	Died	54 days	*Tubercular.
K.	42 days *with contact*	Killed	94 days	*Tubercular.
L.	Alternate exposure as above to room temperature and extreme cold.	Killed	100 days	Animal very well nourished and in excellent condition. A small calcareous nodule at seat of inoculation. Spleen slightly enlarged. A few minute tubercles in lung structure, but rare. Peri-bronchial glands slightly enlarged. Tubercle bacilli found, but with difficulty.
M.	Alternate exposure as above to room temperature and extreme cold.	Killed	100 days	No evidence of tubercle found in animal of English series. In that of French series a few minute tubercles were found in lung structure.

* The emulsion used for inoculation of these two animals was of much greater density than that employed in other cases.

Digitized by Google

The control animals (inoculated from tubes O) died after a period of forty-two, fifty-four, fifty-six, and sixty-three days respectively, the autopsy of these showing marked tuberculosis, affecting almost every organ of the body.

The series of animals of which the autopsy was made at the Pasteur Institute gave results in every way corroborative of those detailed above.

It should be noted :—

1. That the control animals succumbed to the disease at a much earlier date than those inoculated with the exposed material, seven of these latter being still living on the 100th day from the commencement of the experiment. The sole exception to this is guinea-pig No. 13 G., inoculated with material exposed for one week, which died on the thirty-third day.

2. That the time of exposure appeared to make no difference, the animals inoculated with material exposed for forty-two days showing at death tuberculous lesions as pronounced as those in which the material was exposed for the shortest period.

3. That no difference could be traced in the virulence of the material exposed to contact with liquid air.

4. That in animals inoculated with material which had been subjected to alternate exposures it was difficult to find evidence of tubercle. It was only after very careful search that some small tuberculous lesions could be discovered.

To sum up the results of the experiment, it would appear then—

1. That simple exposure to the temperature of liquid air has little or no effect upon the bacillus tuberculosis as far as vitality is concerned.

2. That its virulence is to some degree modified, but not destroyed, by such exposure, even if it be continued for a lengthened period.

3. That length of exposure is not a factor in the question.

4. That actual immersion in liquid air has no special effect upon the organism, nor does it produce results in any way differing from simple exposure to the temperature obtained by it.

5. That successive alternations of extreme cold and normal temperature have a decidedly destructive effect upon the vitality and virulence of the organism.

STEAM-HEATED RAILWAY CARRIAGES AND THE SPREAD OF TUBERCULOSIS.

By A. E. BROOKE HUNT.

In bringing the subject-matter of this paper before the Congress, I must premise my remarks by saying that my sole claim to the indulgence of my hearers lies in the fact that my official duties involve a considerable amount of travelling over practically every railway system in England and Wales, so that I have a very close personal acquaintance with the facts which I bring forward.

Digitized by Google

Ten or twelve years ago, travelling in England in winter was a really pleasurable performance. During the sixteen or eighteen weeks one designates as the winter season one journeyed protected from cold by a rug; with a foot-warmer if one's feet were cold; without the dust and grime of summer, and breathing fresh and comparatively pure air. It is true that occasionally the journey was somewhat cold, but no journey in England can be of very long duration, and it is not often that one experiences a winter which is really severe. It is certainly the exception and not the rule.

Within the period I have mentioned, however, or thereabouts, a change came over the scene. The Westinghouse brake introduced a connection between the engine and every carriage in the train, and, borrowing the idea from countries where the winter is often deadly cold, and where a love of fresh air does not characterise the inhabitants, the railway companies began to connect their carriages by steam-pipes as well as by brake-pipes, and to introduce into each compartment, whether those occupying it desire it or not, a state of heat which makes winter railway travelling a positive terror. There is probably not much need to describe the system; it is, alas! now too well known. Under each seat is placed an iron coil, cylinder, or system of pipes heated by steam conveyed from the engine. The pipes of the different companies vary in size: sometimes they are from three to four inches in diameter; occasionally, as on the Lancashire and Yorkshire Railway, the system is worked by means of cylinders, which must be at least six inches in diameter. The pipes—call them what you will—are always of dull iron, on account no doubt of the radiating properties of this material; occasionally one now finds the pipes enclosed with wood, and so a little of the severity is softened; but whatever the particular system adopted by the railway company may be, in this they all agree—that the heat is *generated at some point under the seat of the carriage.* I do not want to dwell on the general discomfort, the general unhealthiness involved in this system—a system whereby one is heated wholesale, whatever the weather may be. In this connection I have often been amused by the touching belief of railway guards in the thermometers placed in their vans, and, occasionally, in some of the compartments in a train. When one complains of heat which is circulating round one's legs till every pore is freely perspiring, the guard usually retires to the van at the end of the train, where he has a thermometer placed in a position where the outer air plays freely round it. Back he comes in triumph. "The heat that is on, sir, is only 65°!" Or he looks at the thermometer in your compartment, which is so placed as to be a long way from the source of heat, and immediately opposite to the window, which you may have kept open. "See here," he says, "it is only 67°." I have long attempted to study his mind on the point, and believe that he looks on a thermometer much as he does a pressure-gauge. If his gauge can show a pressure which is equal all through the system of pipes with which it is connected, so must it be with a thermometer, no matter where it, or you, may be placed. You may be in a carriage just behind the engine. It does not matter! A thermometer in the van at the end of the train will tell you the heat throughout the system just the same!

Digitized by Google

The point of general unhealthiness, however, cannot be entirely ignored, even when considering the matter in its relation to the spread of a specific disease like tuberculosis. There can be no doubt that by this system the vitality of the body is lowered. In a compartment some 8 feet × 5 feet one has to breathe a superheated and vitiated atmosphere, and this surely must adversely affect the bodily vitality. I use the term "vitiated" advisedly, because it is impossible in a steam-heated carriage to obtain proper ventilation. If you lower the window you certainly enjoy the luxury of cold air blowing upon your head and chest and cooling them, while the lower portion of your body is in a state of perspiration, owing to the heat coming out from under the seat; but even then you are unable to avoid the atmosphere from the floor of the carriage reaching you before it finds its way out at the open window. Some people, however, are afraid of the draught from an open window, and I have been assured by ticket collectors that sometimes, especially on wet and coldish days, when they have to open doors of carriages to collect tickets from the occupants, they are literally made to stagger back on account of the atmosphere which meets them—a Kaffir kraal is probably the only thing comparable to it. Now I venture to think that a bodily system lowered by breathing atmosphere of this nature must be more than ordinarily susceptible to specific disease. I have come across one railway official who was laid up with an attack on the lungs occasioned by having constantly to leave the corridor train of which he was in charge, and be exposed to the cold and draught of the railway station; while more than one guard has complained bitterly of rheumatism induced by the exposure of his limbs, first to the heat from the coils considerately placed beneath the seat in his van, and then to the cold of the platform on which he has to alight; indeed, I am a victim myself, and shall probably suffer to the end of my days from rheumatism brought about by this steam-heating.

These I admit are only instances of the general unhealthiness of the system. How does it bear on danger to the travelling public from infectious diseases? We are given to understand that there is no more fertile source of infection than dust, especially dirty dust. Can anyone tell me of a place which is—area for area—dirtier and dustier than the floor of a railway carriage? Not content with this, which cannot perhaps be entirely avoided, the railway companies place under the seats of carriages their system of steam-pipes, so devised that the heat is generated in close proximity to where the dust lies; it sweeps along the dirty floor; it picks up the dust with which that floor is covered, and carries it, in a way which cannot be avoided, straight into the lungs of those seated in the carriage. If anyone had laid himself out to invent the best possible system of causing the circulation of the dust lying on the floor of railway carriages, could he, short of employing a blast of air, have invented a better system than this for the purpose? And, if this is a perfect system for causing the circulation of dust, I cannot conceive how, by any possibility of means, it could be improved upon as a system for the efficient propagation of disease. No modification of it is to my mind possible. The source of heat must from the very nature of things be placed on the floor,

Digitized by Google

and, placed on the floor, it must cause the circulation of dust and germ-laden air.

But, it may be argued, this is all fancy; there are no more disease germs in a railway carriage than there are anywhere else.

Through the kindness of Professor Hamilton, of Aberdeen University, I have been favoured with a copy of a paper which he read before a Public Health Congress held there last year. In it he explains that he made a number of experimental observations on the dust taken from the floors of railway carriages—both first and third class—running between London and Aberdeen, and Aberdeen and Glasgow. In all, twelve carriages were examined, of which one-half were "smokers." The result was that from samples of dust collected 16·6 per cent., or one-sixth, were found to be contaminated by the bacillus of tubercle. Professor Hamilton—and I cannot do better than quote his exact words—says: "Such a high percentage of instances in which the small quantity of dust removed from railway carriages was found to contain the most deadly of all disease parasites is certainly alarming, to say the least of it, and shows that the danger of infection from this source is extreme, and that persons predisposed to tuberculosis entering such carriages are liable to very great risk of acquiring the disease. If this be the case with tuberculosis," he continues, "the risk of acquiring influenza, and even diphtheria—diseases in which the micro-parasite concerned in their production is voided in the expectoration—must also be very considerable." If this is true of railway carriages in summer, when these experiments were conducted, what must be the risk which is run in winter, when the dust is warmed by the hot air streaming along the floor of the carriage, raised from the floor by the ascending current of warm air, and then inhaled with every breath the unfortunate passenger takes?

We are a peculiar nation. We spend hundreds of thousands of pounds in building sanatoria and endeavouring to cure this terrible disease by means of the "open-air" treatment, and then we allow the railway companies to adopt the most efficient means which could possibly be devised for spreading the disease wholesale.

It is in the hope that by drawing attention to this subject, as I have now been kindly permitted to do, I may rouse others whose voices are more powerful than mine to a sense of the gravity of it, that I have ventured to bring it forward.

Digitized by Google

RÔLE DES EXCRETA BACILLIFÈRES DANS LA PROPAGATION DE LA TUBERCULOSE DES VACHES LAITIÈRES À L'HOMME.

PAR MM.

PROFESSEUR BOINET, *Médecin des Hôpitaux, Correspondant de l'Académie;*

ET

HUON, *Vétérinaire en Chef, Inspecteur-Général des Viandes.*

I. Cette communication complète nos travaux antérieurs * sur la tuberculose observée à Marseille. Elle a pour but d'attirer l'attention sur la fréquence relative de la tuberculose chez les garçons laitiers affectés au service de vacheries étroites, mal aérées, malsaines, renfermant des vaches tuberculeuses. La *transmission de cette tuberculose bovine à l'homme* dans ces conditions spéciales est assez souvent produite par *l'inhalation* des poussières bacillifères provenant du jetage, des mucosités, des expectorations, et même des matières fécales des vaches laitières tuberculeuses. Ravenel † a prouvé expérimentalement que les particules très ténues, projetées au moment de la toux par ces bovidés tuberculeux, sont bacillifères; qu'elles flottent dans l'air; elles peuvent ainsi devenir une source de contagion. Le danger d'infection humaine est, en pratique, limité aux personnes qui vivent en contact prolongé avec ces animaux. À Marseille, ces garçons laitiers y sont d'autant plus exposés qu'ils font un travail excessif, se lèvent avant trois heures du matin, passent leurs journées, et périodiquement la nuit, dans des étables mal conditionnées, où la stagnation du purin et le séjour prolongé du fumier sont la règle. En somme, ces conditions hygiéniques déplorables favorisent singulièrement le développement de la tuberculose chez ces individus surmenées et rapidement anémiés.

II. Il existe, à Marseille, 9000 vaches laitières réparties dans 750 laiteries fournissant 130,000 litres de lait par jour; elles restent de deux à quatre ans dans une stabulation permanente. Les statistiques de nos abattoirs pendant l'année 1900 montrent la *fréquence de la tuberculose chez les vaches laitières :—*

ABATTAGES PENDANT L'ANNÉE 1900.

40,525 bovidés abattus.
- 35,510 *bœufs ou vaches de montagne :—* 4792 cas de tuberculose, soit 14 pour 100.
- 5015 *vaches provenant des vacheries marseillaises :—* 2255 cas de tuberculose, soit 49 pour 100.

---

* Boinet et Huon : "La tuberculose à Marseille, sa prophylaxie" (*Marseille Médical*, 1898); "Transmission de la tuberculose des animaux à l'homme" (*Annales d'Hygiène publique et de médecine légale*, Paris, janvier 1898).

† Ravenel (*University Medic. Magazine*, novembre 1900).

Digitized by Google

Sur 7047 cas de tuberculose observés sur l'espèce bovine, nous n'avons trouvé que 744 cas de généralisation, soit 17 pour 100.

VEAUX	. . . .	29,471	*Pas de Tuberculose.*
MOUTONS et BREBIS	. .	227,408	
AGNEAUX.	. . . .	28,084	
CHEVREAUX	. . . .	233	
PORCS	. . 24,754	.	221 cas de tuberculose, soit 0·88 pour 100.
CHEVAUX.	. 4,594	.	3 cas de tuberculose.
ÂNES.	. . 394	.	néant.

III. La proportion de *tuberculose mammaire* constatée sur les vaches laitières abattues à Marseille donne une moyenne de $2\frac{1}{2}$ pour 100; c'est-à-dire, que sur 2614 cas de tuberculose observée sur des vaches laitières, sortant de nos vacheries marseillaises, nous n'avons trouvé que soixante-dix-quatre cas de tuberculose intéressant les mamelles.

Cependant, des séries d'inoculations intra-péritonéales pratiquées sur le cobaye avec de nombreux échantillons de *lait*, nous ont montré que le lait absorbé par la population marseillaise déterminait de la tuberculose expérimentale dans une proportion de 15·15 pour 100.

Le contraste qui existe entre la rareté de la tuberculose mammaire et un assez grand nombre d'échantillons de lait contenant des bacilles de Koch indique bien que ce liquide est contaminé, pendant et après la traite, par des souillures bacillifères provenant des baves virulentes et des déjections desséchées. Elles peuvent être introduites directement dans le lait par les mains des laitiers qui ne prennent guère des précautions, ou bien elles tombent, plus tard, dans les réservoirs de lait, après avoir flotté dans l'atmosphère de la vacherie où la stabulation permanente des animaux suspects est souvent très prolongée.

IV. Les considérations précédentes prouvent encore que la transmission de la tuberculose aux vaches de la même étable peut se faire aussi par l'inhalation des particules bacillifères pulvérisées et projetées par la toux ou contenues dans les poussières provenant du jetage, de la bave et même des déjections desséchées, lorsqu'il existe des lésions tuberculeuses au niveau de l'intestin des vaches déjà malades.

V. Par contre, l'un de nous * a observé un cas de tuberculose humaine transmise à la vache.

Conclusions :—La propagation de la tuberculose des bovidés à l'homme est à craindre dans les écuries gravement infestées, mal entretenues, obscures, insuffisamment aérées, et lorsque des individus surmenés séjournent longtemps dans ces locaux, renfermant des animaux tuberculeux, y couchent et s'exposent longuement ainsi à toutes ces possibilités de contamination soit par ingestion, soit par inhalations de particules bacillifères que répandent les excreta des vaches tuberculeuses. Il en résulte des conditions analogues à celles que crée une cohabitation habituelle et prolongée avec des malades tuberculeux.

Mesures prophylactiques :—1° Aviser les individus qui séjournent trop

* Huon : " Un cas de tuberculose humaine transmise à une vache " (*Congrès pour l'étude de la tuberculose chez l'homme et chez les animaux*, Quatrième Session, Paris, 1898).

Digitized by Google

longtemps, et couchent dans des étables ainsi contaminées, du réel danger auquel ils s'exposent.

2° Inviter les maires, dans les communes desquels se trouvent des vaches entretenues en stabulation permanente, à faire appliquer la tuberculine dans les vacheries plus ou moins suspectes.

3° Leur conseiller, comme moyen pratique, de prendre un arrêté invitant les laitiers à soumettre toutes leurs vaches à l'épreuve révélatrice de la tuberculine, et comportant les clauses suivantes :—

(*a*) Déclaration du laitier au commissaire de police de son arrondissement, qu'il soumet ses vaches à l'épreuve de la tuberculine, et qu'il confie l'opération à M. X., son vétérinaire, en indiquant le jour de l'opération. Cette déclaration serait immédiatement transmise à la Mairie.

(*b*) Le vétérinaire municipal serait chargé de se mettre en rapport avec le vétérinaire praticien et de se rendre compte de l'opération.

(*c*) Ces deux vétérinaires dresseraient un procès-verbal à M. le Maire, indiquant les résultats de l'opération.

(*d*) Si le procès-verbal porte que toutes les vaches n'ont pas réagi à la tuberculine, c'est-a-dire, sont indemnes de tuberculose, M. le Maire délivrera un certificat sanitaire.

(*e*) Si, par contre, une ou plusieurs vaches ont réagi à la tuberculine et sont, par conséquent, reconnues tuberculeuses, le laitier pourra recevoir le certificat sanitaire, à condition que, dans les quinze jours qui suivent l'injection de la tuberculine, il s'engage à conduire ses vaches à l'abattoir.

(*f*) Un registre tenu par MM. les Commissaires de Police indiquera le nom des laitiers qui ont soumis leurs vaches à l'épreuve de la tuberculine et la date de la délivrance du certificat, avec mission de reprendre le certificat, si au bout d'une année le propriétaire n'a pas fait renouveler l'épreuve de la tuberculine.

(*g*) Les laitiers pourvus d'un certificat sanitaire pourront s'en prévaloir par voie de réclame sur leurs magasins, voitures, bidons. Seuls ils pourront fournir le lait aux administrations publiques—hôpitaux, hospices, lycées, orphelinats. Cette dernière clause existe dans le cahier des charges des hôpitaux de Marseille. Telles sont les principales mesures prophylactiques qui permettraient d'obtenir la suppression du lait tuberculeux et la disparition de la tuberculose dans nos vacheries.

Digitized by Google

INDEX TO VOLUME III.

ACHARD (Dr.), LANNELONGUE (Dr.), et GAILLARD (Dr.), de l'influence comparée des climats et de la résistance individuelle dans la tuberculose expérimentale, 52 ; translation, 56

Acids : relations of tubercle bacilli to other bacteria resistant to acids and to actinomyces (A. Moeller), 485, (W. Bulloch, M.D.) 494

—— transformation of hypo-acid tuberculous soil into hyper-acid arthritic soil (S. Bernheim), 172

Actinomyces : relation of tubercle bacilli to other bacteria resistant to acids and to actinomyces (A. Moeller), 485, (W. Bulloch, M.D.) 494

ADAMI (Prof. J. G., M.D.), discussion on relationship of tubercle bacillus to other bacilli, and on its variations, 503

—— discussion on the infectivity of milk in tuberculous cows, 516

—— discussion on the varieties of tuberculosis (morbid anatomy and histology), 544

Agglutination : agglutinating reaction in cases of pulmonary tuberculosis (P. Horton-Smith, M.D., and H. W. Armit), 151, (Dr. Lydia Rabinowitsch) 153

—— of bacillus of Koch (Drs. Widal and Ravant), 150

Air : devitalised air toxæmia a prime cause of tuberculosis (C. Denison, M.D.), 374

—— (liquid), effect of exposure to liquid air upon vitality and virulence of bacillus tuberculosis (H. Swithinbank), 657

—— (open). *See* "Open air"

Albumin. *See* "Meat albumin"

Alcoholism : relation to tuberculosis (T. N. Kelynack, M.D.), 334

Aldehyde. *See* "Formic aldehyde."

ALLBUTT (Prof. T. Clifford, M.D., F.R.S.), discussion on sanatorium treatment (first opening address), 175

ALLEN (James F., M.D.), on natural immunity from tuberculosis in Natal, South Africa. (Situation, topography, and climate of Natal. Natural immunity from tuberculosis among cattle, monkeys, Europeans, Zulus, Indians. Visitors suffering from pulmonary tuberculosis), 39

Altitude. *See* "High altitude."

AMREIN (O.), über die physiologischen Grundlagen der Hochgebirgskuren und deren Bedeutung für die Tuberculose, 57 ; translation, 67

Anatomy (morbid) of tuberculosis (discussion opened by Prof. Carl Benda), 523

—— of forms of localised tuberculosis (Dr. Clado), 648

ANDERSON (T. McCall, M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 100

ANGIULLI (Gustave), traitement de la tuberculose pulmonaire par les injections hypodermiques de phosphate de chaux ($CaH_4[Ph.O_4]_2H_2O$) ; valeur thérapeutique de la phosphaturie prétuberculaire, expliquée comme phénomène précoce d'intoxication du système nerveux, 423 ; translation of abstract, 422

—— deuxième communication. Traitement de la tuberculose pulmonaire par les injections de phosphate de chaux et la viande crue ou le plasma musculaire, 427

Digitized by Google

Anorexia in the tuberculous: treatment by persodine (persulphate of soda) (J. Hobbs), 452
Antiseptic medicaments: treatment of tuberculosis by direct introduction of antiseptic medicaments to seat of malady through pores of skin by means of static currents of electricity of high and medium tension (Crôtte method) (J. L. Hatch), 471, (A. Salivas) 477, (W. P. Geisse) 479
Antitoxic serums in treatment of tuberculosis (J. Baradat), 429
ARLOING (S.) et COURMONT (Paul), sur le séro-diagnostic de la tuberculose: technique et résultats, 128; translation of abstract, 148
—— —— le séro-diagnostic des tuberculoses dites "chirurgicales," 149; translation, 149
ARMIT (H. W.) and HORTON-SMITH (P., M.D.), the agglutinating reaction in cases of pulmonary tuberculosis, 151
Arthritis: hereditary arthritic forms of tuberculosis (G. E. Papillon), 165
—— transformation of hypo-acid tuberculous soil into hyper-acid arthritic soil (S. Bernheim), 172
AUFFRET (C.), influence du climat sur la tuberculose observée dans l'arsenal maritime de Brest (Finistère), 81; translation of abstract, 83
Bacillus-bearing excreta as factor in communicating tuberculosis from milch-cows to man (Prof. Boinet and M. Huon), 664
Bacillus tuberculosis (of Koch), agglutination of (Drs. Widal and Ravant), 150
—— comparative virulence from human and bovine sources (Prof. M. P. Ravenel), 552, 553–581
—— experiments proving infection of fœtus by (F. F. Friedmann), 632
—— interpretation of lesions caused by (Prof. D. J. Hamilton), 530
—— morphological variations (W. Bulloch, M.D.), 494
—— physiological variations (W. Bulloch, M.D.), 494
—— relations of tubercle bacilli to other bacteria resistant to acids and to actinomyces (A. Moeller), 485
—— relations to other acid-fast bacilli: to ray fungus and other streptothriceae (W. Bulloch, M.D.), 494
—— temperature necessary for killing tubercle bacilli in milk (Prof. D. B. Bang), 592
—— vitality and virulence: effect of exposure to liquid air upon (H. Swithinbank), 657
Bacteriological examination of milk of tuberculous cows (L. Rabinowitsch), 507
BANG (Prof. D. B.), discussion on the infectivity of milk in tuberculous cows, 521
—— some experiments on the temperature necessary for killing tubercle bacilli in milk, 592
BARADAT (J.), sanatorium et villa sanatoria, 264
—— remarks on tuberculosis and its treatment, 401
—— tuberculose et sérums antitoxiques, 429
BARBARY (F.), du cacodylate de gaïacol dans le traitement de la tuberculose, 429
BARDSWELL (Noel D., M.D.), some observations on dietetics in pulmonary tuberculosis, 309
BECHER (Wolf) and LENNHOFF (Rudolf) ("Erholungsstätten"), discussion on sanatorium treatment, 220; translation of abstract, 219
BÉCLÈRE (A.), sur la technique de l'application des rayons de Röntgen au diagnostic de la tuberculose, 276; translation, 278
BENDA (Prof. Carl), opening of discussion on the varieties of tuberculosis (morbid anatomy and histology), 523; reply, 549
—— remarks at close of discussion, 582

Digitized by Google

BERNHEIM (Samuel), de la transformation du terrain tuberculeux hypoacide en terrain arthritique hyperacide, 172
BINET (Maurice) et ROBIN (Albert), la prophylaxie de la tuberculose pulmonaire par la connaissance de son terrain, 157
BOINET (Prof.) et HUON (M.), rôle des excreta bacillifères dans la propagation de la tuberculose des vaches laitières à l'homme, 664
BONNET-LEON (Dr.), sur l'emploi des rayons de Roentgen pour le diagnostic précoce de la tuberculose pulmonaire, 285
BOUSFIELD (E. C.), discussion on the therapeutic and diagnostic value of tuberculin, 105
—— discussion on relationship of tubercle bacillus to other bacilli, and on its variations, 504
—— discussion on the infectivity of milk in tuberculous cows, 519
—— discussion on the varieties of tuberculosis (morbid anatomy and histology), 547
BRAINE-HARTNELL (J. C.), discussion on sanatorium treatment, 226
—— on the value of the rectal temperature in pulmonary tuberculosis, 299
Brest (climate): influence on prevalence of tuberculosis (C. Auffret), 81, 83
BREUILLARD (Ch.), sur un nouveau mode d'excitation de la peau dans le traitement de la tuberculose pulmonaire—le massage pneumatique, 454
British Isles (climate), unsuitability of special types of pulmonary tuberculosis and its complications in (P. S. Hichens, M.B.), 38
Bronchial glands: tuberculosis of tracheal and bronchial glands in miliary tuberculosis of children, and in tuberculous meningitis (P. Haushalter and A. Fruhinsholz), 388, 389
BROWN (William), demonstration of lantern slides on bovine tuberculosis, 592
BUARD (G.), de la séro-réaction comme moyen de diagnostic de la tuberculose; des divers moyens de la pratiquer, 153
BUCK (Arthur H.), the treatment of tuberculosis by urea, 445
BULLOCH (William, M.D.), the morphological and physiological variations of the bacillus tuberculosis and its relations (*a*) to other acid-fast bacilli, (*b*) to the ray fungus and other streptothriceae, 494; reply to discussion, 506
—— discussion on secondary infections and mixed infections in pulmonary tuberculosis, 631
BURTON-FANNING (F. W., M.D.), a report on the results of sanatorium treatment in the United Kingdom, 199
—— discussion on sanatorium treatment, 218
Cacodylate of guaiacol in treatment of pulmonary tuberculosis (F. Barbary), 429
CAMPBELL (Colin), the treatment of phthisis by intra-tracheal injection of large quantities of izal, 406
CAPO (Dr. Espina y), the importance of the X rays as a means for the early diagnosis of tuberculosis, 289
Carriages. *See* "Railway carriages"
Cattle (tuberculosis in): comparative virulence of tubercle bacillus from human and bovine sources (Prof. M. P. Ravenel), 552, 553–581
—— —— lantern-slide demonstrations on bovine tuberculosis (W. Brown), 592
—— —— natural immunity from tuberculosis among cattle in Natal (J. F. Allen, M.D.), 39
—— —— practical value of tuberculin in extirpation of tuberculosis among cattle (L. Rabinowitsch), 507
CERVELLO (Prof. Vincenzo), le traitement de la tuberculose par l'igazol, 416; translation of abstract, 415

Digitized by Google

CHANTEMESSE (Prof.) et CORNIL (Prof.), étude anatomique et expérimentale touchant l'influence de l'alimentation par la viande crue sur l'évolution de la tuberculose expérimentale, 312
Chest-measuring in cases of pulmonary tuberculosis treated at high altitudes (C. Ruedi), 74
Children: raw meat and muscle serum in treatment of pulmonary tuberculosis in (A. Josias and J. C. Roux), 317, 318
—— seaside sanatoria for (Sir H. Weber, M.D.), 234
—— tuberculosis of tracheal and bronchial glands in miliary tuberculosis of children and in tuberculous meningitis (P. Haushalter and A. Fruhinsholz), 388, 389
CLADO (Dr.), étude sur quelques tuberculoses locales: formes cliniques et anatomiques particulières de la localisation tuberculeuse, 648
Classification of popular sanatoria (G. Kuss), 238, 239
Climate: discussion on climatology in relation to treatment of consumption, 1–38
—— grouping of cases for treatment in certain climates (C. Theodore Williams, M.D.), 4
—— influence compared with that of individual resistance in experimental tuberculosis (Drs. Lannelongue, Achard, and Gaillard), 52, 56
—— influence on treatment of consumption (C. Theodore Williams, M.D.), 4
—— of Brest: influence on prevalence of tuberculosis (C. Auffret), 81, 83
—— of British Isles: unsuitability of special types of pulmonary tuberculosis and its complications for treatment in (P. S. Hichens, M.B.), 38
—— of Natal (J. F. Allen, M.D.) 39
Clinical and anatomical forms of localised tuberculosis (Dr. Clado), 648
Convalescent institutions for consumptive out-patients ("Erholungsstätten"), (W. Becher and R. Lenhoff) 219, 220
CORNIL (Prof.) et CHANTEMESSE (Prof.), étude anatomique et expérimentale touchant l'influence de l'alimentation par la viande crue sur l'évolution de la tuberculose expérimentale, 312
COSTA (Jaime R.) [on the use of the Röntgen rays in the diagnosis of pulmonary tuberculosis], 284
COURMONT (Paul) et ARLOING (S.), sur le séro-diagnostic de la tuberculose: technique et résultats, 128; translation of abstract, 148
—— —— le séro-diagnostic des tuberculoses dites "chirurgicales," 149; translation, 149
Cows: bacillus-bearing excreta as factor in communicating tuberculosis from milch-cows to man (Prof. Boinet and M. Huon), 664
—— infectivity of milk of tuberculous cows (L. Rabinowitsch), 495
CRÔTTE (Francisque): reports on the use of his method in the treatment of tuberculosis (J. L. Hatch, M.D.), 471, (A. Salivas) 477, (W. P. Geisse) 479
Cyto-diagnosis in twenty-four cases of sero-fibrinous pleural effusion (Dr. Widal and Dr. Ravant), 150
DELÉPINE (Prof. Sheridan), discussion on destruction of tubercle bacilli in milk, 601
DENISON (Charles, M.D.), discussion on climatology in relation to treatment of consumption, 24
—— discussion on the therapeutic and diagnostic value of tuberculin, 98
—— the uses of tuberculin, 117
—— devitalised air toxæmia—a prime cause of tuberculosis, 374
—— remarks at close of meeting of Medical Section, 484
DENYS (Dr.), discussion on the therapeutic and diagnostic value of tuberculin, 97

Digitized by Google

Destruction of tubercle bacilli in milk, temperature necessary for (Prof. D. B. Bang), 592
Diagnosis. *See* "Cyto-diagnosis," "Roentgen rays," "Serum diagnosis"
Diet in pulmonary tuberculosis (N. D. Bardswell, M.D.), 309
—— *See also* "Meat albumin," "Meat (raw)," "Muscle serum"
Drugs: use in pulmonary tuberculosis (W. R. Huggard, M.D.), 397
DVORÁK (Joh.), über die Errichtung von eigenen Isolirpavillonen für Tuberkulose in allg. offentl. Krankenhäusern, 207
Dystrophy: para-tuberculous hereditary dystrophy (E. Mosny), 166, 167
Electrical currents of high frequency in treatment of tuberculosis (J. A. Rivière), 464
—— of high frequency and high potential in treatment of pulmonary tuberculosis (Chisholm Williams), 467
—— (static) of high and medium tension as means of direct introduction of antiseptic medicaments to seat of malady through pores of skin (J. L. Hatch), 471, (A. Salivas) 477, (W. P. Geisse) 479
England, sanatorium treatment in: results (F. W. Burton-Fanning, M.D.), 199
"Erholungsstätten"—convalescent institutions for consumptive out-patients (W. Becher and R. Lenhoff), 219, 220
Europeans: natural immunity from tuberculosis among Europeans in Natal (J. F. Allen, M.D.), 39
EWART (William, M.D.), on time-saving methods of treatment, with a preliminary note on the "silver treatment" of phthisis by intravenous injections of protargol, 442
EXCHAQUET (Dr.), discussion on climatology in relation to treatment of consumption, 36
Excreta, bacillus-bearing, as factor in communicating tuberculosis from milch-cows to man (Prof. Boinet and M. Huon), 664
Excretion tuberculosis of kidney (Hugh Walsham, M.D.), 552, 583–591
Experimental tuberculosis: influence of climate compared with that of individual resistance in (Drs. Lannelongue, Achard, and Gaillard), 52, 56
—— influence of raw meat diet on (Prof. Cornil and Prof. Chantemesse), 312
Eye, tuberculosis of (A. T. Haight, M.D.), 362
Finsen's method of light treatment applied to lupus vulgaris (demonstration by Dr. J. H. Sequeira), 295
Fœtus: infection by bacilli of tuberculosis, experiments proving (F. F. Friedmann), 632
Formalin: intravenous injections in treatment of pulmonary tuberculosis (R. Maguire, M.D.), 438
Formic aldehyde: inhalation of formic aldehyde an aid in open-air treatment of pulmonary tuberculosis (Chowry Muthu, M.D.), 434
Fossæ (nasal). *See* "Nasal fossæ"
FOULERTON (A. G. R.), the influence of secondary infections in chronic pulmonary phthisis, 604
FOWLER (J. K., M.D.), discussion on sanatorium treatment (second opening address), 186
FRÄNKEL (Professor B.), discussion on the therapeutic and diagnostic value of tuberculin, 97
France. (Sanatoria): work of French medical sanatoria (Dr. Verhaeren), 247
FRANCE (Eric, M.B.), discussion on the therapeutic and diagnostic value of tuberculin, 106
FRIEDMANN (F. F.), on experiments proving the infection of the fœtus by the bacilli of tuberculosis, 632

Digitized by Google

FROTTIER (L.), traitement des tuberculeux par la cure d'air à l'Hôpital Pasteur du Havre, 244
FRUHINSHOLZ (A.) et HAUSHALTER (P.), fréquence et valeur de l'adénopathie tuberculeuse trachéo-bronchique dans la tuberculose miliaire des enfants et dans la méningite tuberculeuse, 389; translation of abstract, 388
GAILLARD (Dr.), LANNELONGUE (Dr.), et ACHARD (Dr.), de l'influence comparée des climats et de la résistance individuelle dans la tuberculose expérimentale, 52; translation, 56
GEISSE (W. P.), report on some experiences in the use of Francisque Crôtte's method in the treatment of consumption, 479
Glands. *See* "Bronchial glands"; "Tracheal glands"
GRAM (Prof. Christian), discussion on climatology in relation to treatment of consumption, 32
Guaiacol (cacodylate of). *See* "Cacodylate of guaiacol"
GUETSCHEL (Dr.) et JONNART (Dr. P.), résultats de la cure d'altitude au sanatorium d'Hauteville (Ain), France (with translation), 51
HABERSHON (S. H., M.D.), discussion on the varieties of tuberculosis (morbid anatomy and histology), 536
Habitat of pulmonary tuberculosis: acquaintance with it a guide to prophylaxis (A. Robin and M. Binet), 157
HAIGHT (Allen T., M.D.), tuberculosis of the eye, 362
HAMBLETON (G. W.), discussion on sanatorium treatment, 231
HAMILTON (Prof. D. J.), discussion on the infectivity of milk in tuberculous cows, 518
—— on the interpretation of the lesions caused by the bacillus tuberculosis, 530; reply, 548
HAMILTON (W. M., M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 105
HATCH (J. Leffingwell, M.D.), a report on the treatment of tuberculosis by direct introduction of antiseptic medicaments to the seat of the malady through the pores of the skin by means of static currents of electricity of high and medium tension (Crôtte method), 471
HAUSHALTER (P.) et FRUHINSHOLZ (A.), fréquence et valeur de l'adénopathie tuberculeuse trachéo-bronchique dans la tuberculose miliaire des enfants et dans la méningite tuberculeuse, 389; translation of abstract, 388
Hauteville: results of high-altitude treatment at sanatorium of Hauteville (Ain), France (Dr. P. Jonnart and Dr. Guetschel), 51
Havre: open-air treatment at Pasteur hospital, Havre (L. Frottier), 244
Heart, tuberculosis of (H. N. Heineman, M.D.), 344
HEINEMAN (H. Newton, M.D.), tuberculosis of the heart, 344
Heredity: hereditary arthritic forms of tuberculosis (G. E. Papillon), 165
—— in relation to immunity and selective activity in tuberculosis (H. M. King, M.D.), 641
—— para-tuberculous hereditary dystrophy (E. Mosny), 166, 167
HERON (G. A., M.D.), opening of combined discussion on the therapeutic and diagnostic value of tuberculin in human tuberculosis, 84; reply, 111
HICHENS (P. S., M.B.), a query as to whether any special types of pulmonary tuberculosis and its complications are unsuitable for treatment in the climate of the British Isles, 38
High-altitude treatment, chest-measuring in cases of pulmonary tuberculosis under (C. Ruedi), 74
—— —— physiological principles and their importance with respect to tuberculosis (O. Amrein), 57, 67

Digitized by Google

High-altitude treatment: results at sanatorium of Hauteville (Ain), France (Dr. P. Jonnart and Dr. Guetschel), 51
—— —— statistics and results in pulmonary tuberculosis (Th. Stephani), 77, 79
High-frequency electrical currents in treatment of tuberculosis (J. A. Rivière), 464
—— —— and high-potential electrical currents in treatment of pulmonary tuberculosis (Chisholm Williams), 467
Histology of tuberculosis (discussion opened by Prof. Carl Benda), 523
HOBBS (J.), treatment of anorexia in the tuberculous by persodine (persulphate of soda), 452
HOBHOUSE (E., M.D.), discussion on climatology in relation to treatment of consumption, 35
HORTON-SMITH (P., M.D.) and ARMIT (H. W.), the agglutinating reaction in cases of pulmonary tuberculosis, 151
Hospitals (public general), establishment of isolation blocks for tuberculous cases in (Joh. Dvorák), 209
HUGGARD (W. R., M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 110
—— discussion on sanatorium treatment, 224
—— the use of drugs in pulmonary tuberculosis, 397
HUNT (A. E. Brooke), steam-heated railway carriages and the spread of tuberculosis, 660
HUON (M.) et BOINET (Prof.), rôle des excreta bacillifères dans la propagation de la tuberculose des vaches laitières à l'homme, 664
Hypodermic injections of phosphate of lime in treatment of pulmonary tuberculosis (G. Angiulli), 422, 423
—— —— —— —— —— —— with raw meat diet in treatment of pulmonary tuberculosis (G. Angiulli), 427
Igazol in treatment of tuberculosis (Prof. V. Cervello), 415, 416
Immunity: heredity in relation to immunity and selective activity in tuberculosis (H. M. King, M.D.), 641
—— in relation to serum treatment of tuberculosis (T. Yabé), 635
—— (natural) from tuberculosis among cattle, monkeys, Europeans, Zulus, Indians, in Natal (J. F. Allen, M.D.), 39
Individual resistance: influence compared with that of climate in experimental tuberculosis (Drs. Lannelongue, Achard, and Gaillard), 52, 56
Infection of fœtus by bacilli of tuberculosis, experiments proving (F. F. Friedmann), 632
Infections (mixed) in pulmonary tuberculosis (Prof. W. Ophüls), 614
—— (secondary): influence in chronic pulmonary phthisis (A. G. R. Foulerton), 604
Infectivity of milk of tuberculous cows (L. Rabinowitsch), 507
—— —— —— of tuberculous cows reacting to tuberculin test (E. J. McWeeney, M.D.), 514
Inhalation of formic aldehyde an aid in open-air treatment of pulmonary tuberculosis (Chowry Muthu, M.D.), 434
Institutions, treatment in (E. Symes Thompson, M.D.), 249
Intoxication of nervous system, pretubercular phosphaturia early sign of (G. Angiulli), 422, 423
Intravenous injections of formalin in treatment of pulmonary tuberculosis (R. Maguire, M.D.), 438
—— —— —— of protargol (silver treatment of phthisis) (Wm. Ewart, M.D.), 442
Ireland, sanatorium treatment in: results (F. W. Burton-Fanning, M.D.), 199

Digitized by Google

Isolation blocks : establishment for tuberculous cases in public general hospitals (Joh. Dvorák), 209
Izal : intra-tracheal injection of large quantities of izal in treatment of phthisis (C. Campbell), 406
JANEWAY (Prof. E. G.), discussion on sanatorium treatment, 211
JONNART (Dr. P.) and GUETSCHEL (Dr.), résultats de la cure d'altitude au sanatorium d'Hauteville (Ain), France (with translation), 51
JOSIAS (Albert) et ROUX (Jean Ch.), résultats du traitement de la tuberculose pulmonaire chez les enfants par le suc musculaire et la viande crue, 318; translation of abstract, 317
KELYNACK (T. N.), the relation of alcoholism to tuberculosis, 334
Kidney, excretion tuberculosis of (Hugh Walsham, M.D.), 552, 583–591
KING (Herbert Maxon, M.D.), a study in heredity : in its relation to immunity and selective activity in tuberculosis, 641
KINGWELL (H. A.), discussion on comparative virulence of tubercle bacillus from human and bovine sources, 602
KNOPF (S. A., M.D.), discussion on sanatorium treatment, 214
KOCH (Prof. Karl), discussion on the therapeutic and diagnostic value of tuberculin, 93 ; reply, 114, 115
—— *See* "Bacillus tuberculosis" (of Koch)
KOSSEL (Prof.), discussion on relationship of tubercle bacillus to other bacilli, and on its variations, 505
—— discussion on destruction of tubercle bacilli in milk, 599
KUSS (G.), les sanatoriums populaires de répartition, 239 ; translation of abstract, 238
KUTTNER (A.), Larynxtuberkulose und Gravidität, 353 ; translation of abstract, 355
LANNELONGUE (Dr.), ACHARD (Dr.), et GAILLARD (Dr.), de l'influence comparée des climats et de la résistance individuelle dans la tuberculose expérimentale, 52 ; translation, 56
Lantern-slide demonstration on bovine tuberculosis (W. Brown), 592
Laryngitis (tuberculous) : principles of treatment (StC. Thomson, M.D.), 356
Larynx, tuberculosis of, in relation to pregnancy (A. Kuttner), 353, 355
—— —— —— part played by nasal fossæ in prophylaxis and treatment of (M. Mignon), 390, 391
LASALLE (Dr.) [on marine sanatoria], 263
Lesions caused by bacillus tuberculosis, interpretation of (Prof. D. J. Hamilton), 530
LENNHOFF (Rudolf) and BECHER (Wolf), "Erholungsstätten" (discussion on sanatorium treatment), 220 ; translation of abstract, 219
Light treatment (Finsen's method) applied to lupus vulgaris (demonstration by Dr. J. H. Sequeira), 295
Lime (phosphate of). *See* "Phosphate of lime"
Liquid air. *See* "Air (liquid)"
Localisation (tuberculous) : clinical and anatomical forms (Dr. Clado), 648
Lupus vulgaris : treatment by Finsen's method and by X rays (demonstration by Dr. J. H. Sequeira), 295
MAGUIRE (R., M.D.), treatment of pulmonary tuberculosis by means of formalin intravenous injections, 438
Maltese Islands : tuberculosis in (Them. Zammit), 74
Marine sanatoria (Dr. Lasalle), 263
—— for children in prevention and treatment of scrofulous complaints (Sir H. Weber, M.D.), 234

Digitized by Google

MARMOREK (A.), discussion on relationship of tubercle bacillus to other bacilli, and on its variations, 504
—— discussion on comparative virulence of tubercle bacillus from human and bovine sources, 602
Massage (pneumatic) in treatment of pulmonary tuberculosis (Ch. Breuillard), 454
MCWEENEY (Prof. E. J., M.D.), discussion on relationship of tubercle bacillus to other bacilli, and on its variations, 504
—— on the infectivity of the milk of cows that react to the tuberculin test in the absence of clinical evidence of tuberculosis, 514; reply, 521
—— discussion on the varieties of tuberculosis (morbid anatomy and histology), 541; remarks at close of discussion, 582
—— discussion on destruction of tubercle bacilli in milk, 601
Meat: resolution in favour of prevention of spread of tuberculosis by meat and milk, 603
Meat (raw): influence of raw meat diet on experimental tuberculosis (Prof. Cornil and Prof. Chantemesse), 312
—— —— raw meat diet with injections of phosphate of lime in treatment of pulmonary tuberculosis (G. Angiulli), 427
—— —— treatment of pulmonary tuberculosis in children by raw meat and muscle serum (A. Josias and J. C. Roux), 317, 318
Meat albumin dietary in treatment of tuberculosis (F. W. Forbes Ross, M.D.), 330
Meningitis (tuberculous): tuberculosis of tracheal and bronchial glands in tuberculous meningitis (P. Haushalter and A. Fruhinsholz), 388, 389
MIGNON (Maurice), difficulté en interprétation des images en radiologie, 294
—— le rôle des fosses nasales dans la prophylaxie et le traitement de la tuberculose pulmonaire et laryngée, 391; translation of abstract, 390
Miliary tuberculosis of children: tuberculosis of tracheal and bronchial glands in (P. Haushalter and A. Fruhinsholz), 388, 389
Milk: bacillus-bearing excreta as factor in communicating tuberculosis from milch-cows to man (Prof. Boinet and M. Huon), 664
—— of tuberculous cows: bacteriological examination (L. Rabinowitsch), 507
—— —— infectivity of (L. Rabinowitsch), 507, (E. J. McWeeney, M.D.) 514
—— resolution in favour of prevention of spread of tuberculosis by meat and milk, 603
—— temperature necessary for killing tubercle bacilli in milk (Prof. D. B. Bang), 592
Mixed infections in pulmonary tuberculosis (Prof. W. Ophüls), 614
MOELLER (Alfred), discussion on the therapeutic and diagnostic value of tuberculin, 108
—— on the relations of tubercle bacilli to other bacteria resistant to acids and to actinomyces, 485; reply to discussion, 506
Monkeys: natural immunity from tuberculosis among monkeys in Natal (J. F. Allen, M.D.), 39
MOORE (Sir John, M.D.), discussion on climatology in relation to treatment of consumption, 32
—— discussion on treatment in institutions and open-air nursing homes, 259
—— remarks at close of meeting of Medical Section, 483
Morbid anatomy. *See* "Anatomy (morbid)"
MORIN (F.), discussion on sanatorium treatment, 229
Morphological variations of bacillus tuberculosis (W. Bulloch, M.D.), 494
MOSNY (E.), l'hérédo-dystrophie paratuberculeuse, 167; translation, 166
MUIR (Prof. Robert), discussion on the varieties of tuberculosis (morbid anatomy and histology), 545

Digitized by Google

Muscle serum : treatment of pulmonary tuberculosis in children by raw meat and muscle serum (A. Josias and J. C. Roux), 317, 318
MUTHU (Chowry, M.D.), the inhalation of formic aldehyde as an aid in the open-air treatment of pulmonary tuberculosis, 434
Nasal fossæ in relation to prophylaxis and treatment of pulmonary and laryngeal tuberculosis (M. Mignon), 390, 391
Natal : natural immunity from tuberculosis among cattle, monkeys, Europeans, Zulus, Indians (J. F. Allen, M.D.), 39
—— situation, topography, and climate (J. F. Allen, M.D.), 39
—— visitors to, suffering from pulmonary tuberculosis (J. F. Allen, M.D.), 39
Nervous system, pretubercular phosphaturia early sign of intoxication of (G. Angiulli), 422, 423
NIXON (Sir Christopher, M.D.), discussion on the varieties of tuberculosis (morbid anatomy and histology), 543
NOCARD (M.), discussion on the infectivity of milk in tuberculous cows, 515
NOLEN (Professor C.), discussion on the therapeutic and diagnostic value of tuberculin, 111
Nursing homes: treatment in open-air nursing homes (E. Symes Thompson, M.D.), 249
Open-air nursing homes, treatment in (E. Symes Thompson, M.D.), 249
Open-air treatment at the Pasteur hospital, Havre (L. Frottier), 244
—— —— treatment of pulmonary tuberculosis, inhalation of formic aldehyde as aid to (Chowry Muthu, M.D.), 434
OPHÜLS (Prof. W.), mixed infections in pulmonary tuberculosis, 614
OSLER (Prof. William, M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 96
—— discussion on the varieties of tuberculosis (morbid anatomy and histology), 539
OTIS (Edward, M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 110
PAPILLON (G. E.), the hereditary arthritic forms of tuberculosis, 165
PARSONS (Alfred R., M.D.), discussion on sanatorium treatment, 227
Pasteur hospital, Havre : open-air treatment at (L. Frottier), 244
Persodine. *See* "Persulphate of soda."
Persulphate of soda : treatment of anorexia in the tuberculous by persodine (persulphate of soda) (J. Hobbs), 452
PHILIP (R. W., M.D.), discussion on sanatorium treatment (third opening address), 190
Phosphate of lime : hypodermic injections in treatment of pulmonary tuberculosis (G. Angiulli), 422, 423
—— —— with raw meat diet in treatment of pulmonary tuberculosis (G. Angiulli), 427
Phosphaturia (pretubercular), early sign of intoxication of nervous system (G. Angiulli), 422, 423
Physiological principles of high-altitude treatment in tuberculosis (O. Amrein), 57, 67
—— variations of bacillus tuberculosis (W. Bulloch, M.D.), 494
Pleura : cyto-diagnosis in twenty-four cases of sero-fibrinous pleural effusion (Dr. Widal and Dr. Ravant), 150
Pneumatic massage in treatment of pulmonary tuberculosis (Ch. Breuillard), 454
POWELL (Sir R. Douglas, Bt., K.C.V.O.), address as President of the Medical Section, 1
—— discussion on the therapeutic and diagnostic value of tuberculin, 96
—— discussion on sanatorium treatment, 205, 232

Digitized by Google

Pregnancy, tuberculosis of larynx in relation to (A. Kuttner), 353, 355
Prophylaxis against pulmonary tuberculosis through acquaintance with its habitat (A. Robin and M. Binet), 157
Protargol : intravenous injections (silver treatment of phthisis) (Wm. Ewart, M.D.), 442
QUARRIER (William), discussion on climatology in relation to treatment of consumption, 34
RABINOWITSCH (Dr. Lydia), the agglutinating reaction in cases of pulmonary tuberculosis, 153
—— on the infectivity of the milk of tuberculous cows, the bacteriological diagnosis, and the practical value of tuberculin in the extirpation of tuberculosis among cattle, 507
Railway carriages : steam-heated railway carriages in relation to spread of tuberculosis (A. E. B. Hunt), 660
RAVANT (Dr.) et WIDAL (Dr.), recherches sur l'agglutination du bacille de Koch et le cyto-diagnostic dans vingt-quatre cas d'épanchement séro-fibrineuse de la plèvre, 150
RAVENEL (Prof. M. P.), discussion on the infectivity of milk in tuberculous cows, 507
—— the comparative virulence of the tubercle bacillus from human and bovine sources, 552, 553–581 ; reply, 603
Ray fungus. *See* " Actinomyces "
Rectum, temperature of : value in pulmonary tuberculosis (J. C. Braine-Hartnell), 299
Resistance (individual) : influence compared with that of climate in experimental tuberculosis (Drs. Lannelongue, Achard, and Gaillard), 52, 56
RIVIÈRE (J. A.), treatment of tuberculosis by high-frequency currents, 464
ROBIN (Albert) et BINET (Maurice), la prophylaxie de la tuberculose pulmonaire par la connaissance de son terrain, 157
Roentgen rays, diagnosis of pulmonary tuberculosis by (H. Walsham, M.D.), 267
—— —— —— —— —— —— (J. R. Costa), 284
—— —— —— —— —— —— technique (A. Béclère), 276, 278
—— —— —— —— early diagnosis of pulmonary tuberculosis by (Dr. Bonnet-Leon), 285
—— —— —— —— —— —— (Dr. Espina y Capo), 289
—— difficulty in distinguishing radioscopic objects (Dr. M. Mignon), 294
—— lupus vulgaris treated by (demonstration by Dr. J. H. Sequeira), 295
ROSENTHAL (Dr.), discussion on sanatorium treatment, 216
ROSS (F. W. Forbes, M.D.), meat albumin dietary in the treatment of tuberculosis, 330
ROUX (Jean Ch.), résultats du traitement de la tuberculose pulmonaire chez les enfants par le suc musculaire et la viande crue, 318 ; translation of abstract, 317
RUATA (Carlo), discussion on comparative virulence of tubercle bacillus from human and bovine sources, 598, 603
RUEDI (Carl), personal experience in chest-measuring in cases of pulmonary tuberculosis treated at high altitudes, 74
SALIVAS (Albert), report on the treatment of tuberculosis by Francisque Crôtte's method, 477
Sanatoria (treatment by) : discussion on sanatorium treatment (opening addresses by T. Clifford Allbutt, M.D., J. K. Fowler, M.D., and R. W. Philip, M.D.), 175, 186, 190
—— —— results in United Kingdom (F. W. Burton-Fanning, M.D.), 199
—— (closed) contrasted with open or home sanatoria (sanatorium et villas sanatoria) (Dr. J. Baradat), 264

Digitized by Google

Sanatoria (French) : work of French medical sanatoria (Dr. Verhaeren), 247
—— (marine) (Dr. Lasalle), 263
—— —— seaside sanatoria for children in prevention and treatment of scrofulous complaints (Sir H. Weber, M.D.), 234
—— (open or home) contrasted with closed sanatoria (sanatorium et villas sanatoria) (Dr. J. Baradat), 262
—— (popular) classification of (G. Kuss), 238
Sanatorium of Hauteville, results of high-altitude treatment at (Dr. P. Jonnart and Dr. Guetschel), 51
SANDWITH (F. M.), discussion on climatology in relation to treatment of consumption, 33
VON SCHRÖTTER (Professor), discussion on sanatorium treatment, 205
Scotland, sanatorium treatment in : results (F. W. Burton-Fanning, M.D.), 199
Scrofula, prevention of, and treatment in children by means of seaside sanatoria (Sir H. Weber, M.D.), 234
Seaside sanatoria. *See* "Marine sanatoria," "Sanatoria (marine)"
Secondary infections: influence in chronic pulmonary phthisis (A. G. R. Foulerton), 604
Selective activity : heredity in relation to immunity and selective activity in tuberculosis (H. M. King, M.D.), 641
SEQUEIRA (J. H., M.D.), demonstration on cases of lupus vulgaris treated by Finsen's method and by the X rays ; also results of other forms of treatment, 295
Serum (antitoxic) : tuberculosis treated by antitoxic serums (J. Baradat), 429
Serum-diagnosis of tuberculosis in surgical cases (S. Arloing and P. Courmont), 149
—— —— technique and results (S. Arloing and P. Courmont), 128, 148
—— —— various methods of practising it (G. Buard), 153
—— *See also* "Agglutination"
Serum treatment of tuberculosis, immunity in relation to (T. Yabé), 635
—— *See also* "Muscle serum"
"Silver treatment" of phthisis by intravenous injections of protargol (Wm. Ewart, M.D.), 442
SINCLAIR (Howard, M.D.), discussion on sanatorium treatment, 227
Skin : direct introduction of antiseptic medicaments to seat of malady through pores of skin by means of static currents of electricity of high and medium tension (Crôtte method), (J. L. Hatch), 471, (A. Salivas) 477, (W. P. Geisse) 479
—— new method of stimulation in treatment of pulmonary tuberculosis—pneumatic massage (Ch. Breuillard), 454
SNOW (W. Vicary, M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 105
—— discussion on sanatorium treatment, 215
Soda (persulphate of). *See* "Persulphate of soda"
Soil of tuberculosis : transformation of hypo-acid tuberculous soil into hyper-acid arthritic soil (S. Bernheim), 172
SQUIRE (J. Edward, M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 108
—— discussion on treatment in institutions and open-air nursing homes, 261
Statistics and results of high-altitude treatment in pulmonary tuberculosis (Th. Stephani), 77, 79
Steam-heated railway carriages in relation to spread of tuberculosis (A. E. B. Hunt), 660

Digitized by Google

STEPHANI (Th.), statistique et résultats de la cure d'altitude dans la tuberculose pulmonaire, 77 ; translation, 79
STONE (A. K., M.D.), discussion on the therapeutic and diagnostic value of tuberculin, 106
—— discussion on sanatorium treatment, 230
Streptothriceae : relations of bacillus tuberculosis to ray fungus and other streptothriceae (W. Bulloch, M.D.), 494
Surgical cases of tuberculosis, serum diagnosis in (S. Arloing and P. Courmont), 149
SWITHINBANK (Harold), effect of exposure to liquid air upon the vitality and virulence of bacillus tuberculosis, 657
Technique of serum diagnosis in tuberculosis (S. Arloing and P. Courmont), 128, 148
Temperature necessary for killing tubercle bacilli in milk (Prof. D. B. Bang), 592
—— (rectal) : value in pulmonary tuberculosis (J. C. Braine-Hartnell), 299
THOMPSON (E. Symes, M.D.), treatment in institutions and in open-air nursing homes, 249
THOMSON (StClair, M.D.), the principles of treatment of tuberculous laryngitis, 356
Time-saving methods of treatment of phthisis (Wm. Ewart, M.D.), 442
Topography of Natal (J. F. Allen, M.D.), 39
Toxæmia : devitalised air toxæmia—a prime cause of tuberculosis (C. Denison, M.D.), 374
Trachea : intra-tracheal injection of large quantities of izal in treatment of phthisis (C. Campbell), 406
Tracheal glands : tuberculosis of tracheal and bronchial glands in miliary tuberculosis of children and in tuberculous meningitis (P. Haushalter and A. Fruhinsholz), 388, 389
Tuberculin : infectivity of milk of cows reacting to tuberculin test in absence of clinical evidence of tuberculosis (E. J. McWeeney, M.D.), 514
—— practical value in extirpation of tuberculosis among cattle (L. Rabinowitsch), 507
—— therapeutic and diagnostic value in treatment of tuberculosis (G. A. Heron, M.D.), 84
—— uses of (C. Denison, M.D.), 117
United Kingdom. *See* "England," "Ireland," "Scotland"
Urea, treatment of tuberculosis by (A. H. Buck), 445
Varieties of tuberculosis (morbid anatomy and histology) (discussion opened by Prof. Carl Benda), 523
VERHAEREN (Dr.), l'œuvre des sanatoriums médicaux français, 247
"Villas sanatoria" (J. Baradat), 264
Virulence of bacillus tuberculosis (comparative) from human and bovine sources (Prof. M. P. Ravenel), 552, 553–581
—— effect of exposure to liquid air upon (H. Swithinbank), 657
Visitors to Natal suffering from pulmonary tuberculosis (J. F. Allen, M.D.), 39
Vitality of bacillus tuberculosis, effect of exposure to liquid air upon (H. Swithinbank), 657
VIVANT (J. E.), discussion on the therapeutic and diagnostic value of tuberculin, 107
WALCH (C. N. Crosby, M.B.), discussion on climatology in relation to treatment of consumption, 35
WALSHAM (Hugh, M.D.), on the use of the Röntgen rays in the diagnosis of pulmonary tuberculosis, 267
—— excretion tuberculosis of the kidney, 552, 583–591

Digitized by Google

Digitized by Google